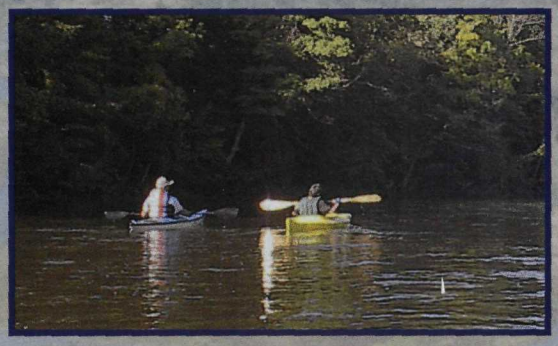
Stage I Assessment Report, Volume 1 — Injury Assessment: Kalamazoo River Environment

Final Report



Michigan Department of Environmental Quality,
Michigan Attorney General,
U.S. Fish and Wildlife Service,
and
National Oceanic and Atmospheric Administration

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Contents

Figures

Tables

1. Introduction

The Director of the Michigan Department of Environmental Quality (MDEQ), the Attorney General of the State of Michigan, the U.S. Secretary of the Interior as represented by the Regional Director of the U.S. Fish and Wildlife Service (U.S. FWS), and the U.S. Secretary of Commerce as represented by the National Oceanic and Atmospheric Administration (NOAA) (collectively referred to as the Trustees), are in the process of assessing damages for injuries to natural resources in the Kalamazoo River Environment (KRE) resulting from the release of hazardous substances into the KRE.[1]

The Comprehensive Environmental Response, Compensation, and Liability Act of 1980 (CERCLA), as amended, 42 U.S.C. §§ 9601-75, and the Federal Water Pollution Control Act (Clean Water Act, or CWA), 33 U.S.C. §§ 1251-1387, provide authority for the Trustees to seek such damages. Additionally, the State Trustees have authority to seek damages for the full value of the injuries to natural resources pursuant to Section 20126a(1)(c) of Part 201, Environmental Remediation, of the Natural Resources and Environmental Protection Act (NREPA), MCL § 324.20126, as well as Section 3115(2) of Part 31, Water Resources Protection, of NREPA, MCL § 324.3115(2).

1.1 Natural Resource Damage Assessment Process

The Trustees have decided to conduct the natural resource damage assessment (NRDA) for the KRE in stages. The first stage of the assessment entails the development of initial conclusions regarding the types and magnitudes of injury and damages resulting from hazardous substance releases into the KRE. The Stage I Assessment is deemed preliminary because future injuries are dependent on the remedial actions that will be implemented at the site,[2] and the response agencies have not yet made decisions regarding remediation. The Stage I Assessment is based primarily on data known to and available to the Trustees through approximately 2003 and on additional information the Trustees were aware of as of the date of this report. In the event the Trustees become aware of new or additional information pertinent to the assessment, including information regarding other hazardous substances or releases, the Trustees may revise the results

1. On September 29, 2004, the Michigan Department of Natural Resource (MDNR) was designated to serve as a natural resource Trustee along with the MDEQ and the Attorney General of the State of Michigan. As of the date of publication of this report, efforts are underway to include the MDNR as a member of the Trustee Council to assure the coordination of future NRDA activities.

2. The term "site" as used in this document refers to the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site, and any additional areas addressed by the remedial investigation (see Section 1.3).

of the Stage I Assessment or address such information in subsequent stages of the assessment. The results of the Stage I Assessment will be used by the Trustees to help define any additional focused work that could be conducted in the next stage and, if appropriate, to help evaluate any potential settlement options. If deemed necessary by the Trustees, a more detailed Stage II Assessment may be conducted, in which further studies may address uncertainties identified in the Stage I Assessment.

This document presents the Stage I Injury Assessment by the NRDA Trustees. A companion document presents the Trustees' Stage I Economic Assessment Report (Michigan Department of Environmental Quality et al., 2005). The Stage I Assessment is the third step in the NRDA process and follows the KRE Preassessment Screen prepared by the Trustees in May 2000 (Michigan Department of Environmental Quality et al., 2000a) and the Stage I Assessment Plan prepared by the Trustees in November 2000 (Michigan Department of Environmental Quality et al., 2000b). Based on the conclusions of the Preassessment Screen, the Trustees determined that there is a reasonable probability of making a successful natural resource damages claim, and that they would proceed with the preparation of a Stage I Assessment Plan. The Stage I Assessment Plan described the approach and methods to be used in the Stage I Assessment. This Stage I Assessment was prepared in accordance with the U.S. Department of the Interior (DOI, or the Department) NRDA regulations as set forth at 43 C.F.R. Part 11.[3]

1.2 Public Review and Comment

While the Stage I Assessment Report is not subject to a public comment period under state or federal law, the Trustees recognize the benefits of public involvement. Consequently, the Trustees will consider written comments received by April 15, 2005 when planning and undertaking additional assessment activities.

Written comments may be submitted to:

Nanette D. Leemon
Michigan Department of Environmental Quality
Compliance and Enforcement Section
Remediation and Redevelopment Division
P.O. Box 30426
Lansing, Michigan 48909

3. 43 C.F.R. Part 11 regulations were authored by the DOI, and are referred to as the DOI regulations in this document. Use of these regulations is not required. However, they must be used in order for a NRDA to be accorded the evidentiary status of a rebuttable presumption. 43 C.F.R. § 11.11.

Information disseminated by federal agencies to the public after October 1, 2002 is subject to information quality guidelines developed by each agency pursuant to Section 515 of Public Law 106-554 that are intended to ensure and maximize the quality of such information (i.e., the objectivity, utility and integrity of such information). This Stage I Assessment Report is an information product covered by information quality guidelines established by NOAA and DOI for this purpose. The information contained herein complies with applicable guidelines.

1.3 Background Information on the Assessment Area

Polychlorinated biphenyls (PCBs) have been released into the environment in the vicinity of Kalamazoo, Michigan (Figure 1.1). The primary industrial activity associated with the PCB releases into the KRE was the recycling of carbonless copy paper at several area paper mills. Carbonless copy paper manufactured from 1954 to 1971 contained PCBs (Appleton Papers, 1987). Waste from the recycling process conducted at Kalamazoo area paper companies also contained PCBs, and the waste was disposed of by several methods which resulted in releases of PCBs into the environment.

The Allied Paper, Inc./Portage Creek/Kalamazoo River National Priorities List (NPL) site is located in Kalamazoo and Allegan counties, Michigan, and includes multiple sources of PCBs. Industrial and potentially other activities in the Kalamazoo area released PCBs into the environment, including Portage Creek and the Kalamazoo River. PCBs have contaminated sediments, the water column and biota in and adjacent to an 80-mile stretch of the river. This site was included on the NPL on August 30, 1990. The U.S. Environmental Protection Agency (EPA) and Michigan describe the site as follows: (1) an Allied Paper, Inc. property in Kalamazoo, Kalamazoo County, Michigan; (2) a 3-mile stretch of Portage Creek from Cork Street just above the Bryant Mill Pond in the city of Kalamazoo to where the Creek meets the Kalamazoo River; and (3) an approximately 80-mile stretch of the Kalamazoo River, from Morrow Dam to Lake Michigan, with adjacent floodplains, wetlands, and in-stream sediments. The site currently includes several paper residual disposal areas[4] and paper mill properties[5] (see Figure 1.2 and Section 2.2, infra). At this time the site includes five cleanup projects and/or

4. The paper residual disposal areas include the Monarch historical residuals dewatering lagoon (or "HRDL"), the Bryant HRDL, the former Bryant Mill Pond, the King Highway Landfill, the A-Site Disposal Area, the Willow Boulevard Disposal Area, the 12th Street Landfill, and the Fort James Disposal Area.

5. The paper mill properties include the Allied Paper, Inc. mills including Monarch, Bryant and King; the Georgia-Pacific mills; the Simpson-Plainwell Mill; and the Ft. James Paperboard Mill and KVP Mill.

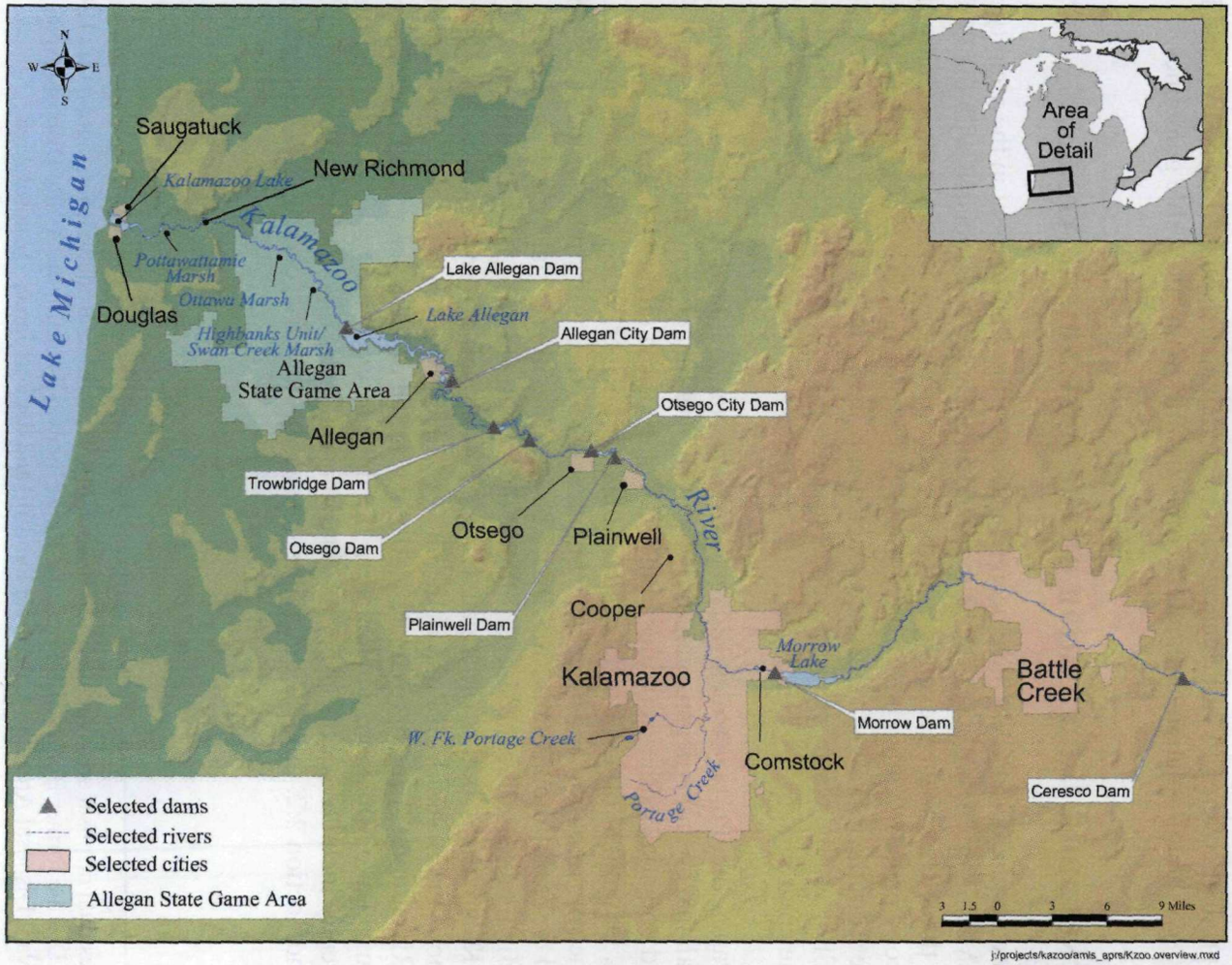

Figure 1.1. Key features of the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site and surrounding environment.

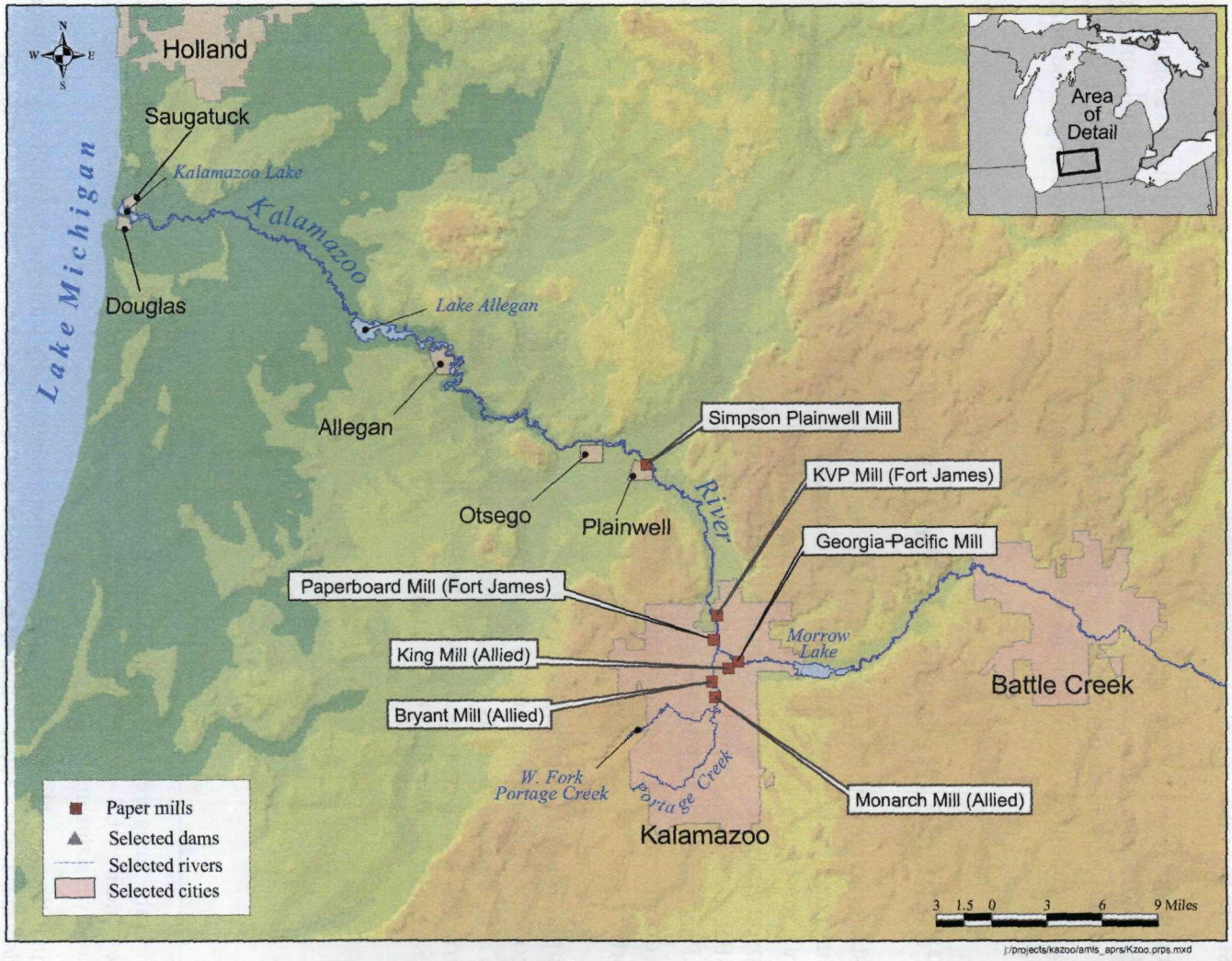

Figure 1.2. Location of current and former PRP paper mill facilities.

operable units.[6] The Superfund Remedial Investigation (RI) for the Portage Creek and Kalamazoo River operable unit includes the Kalamazoo River area from Battle Creek downstream to the mouth of the Kalamazoo River at Lake Michigan about 80 river miles northwest and the three miles of Portage Creek.

In this document, the Trustees are using the term KRE to represent the entire assessment area. The KRE encompasses the area currently included in the RIs for the site's operable units along with any area where hazardous substances released at or from the site have come to be located or where natural resources or the services they provide may have been affected by these hazardous substances.

1.4 Potentially Responsible Parties

The Trustees have identified the following potentially responsible parties (PRPs): Allied Paper, Inc. and its parent company, Millennium Holdings, Inc. (now owned by Lyondell Chemical Company); the Georgia-Pacific Corporation; Plainwell Inc. (successor to Plainwell Paper Inc. and Simpson Plainwell Paper Company); and the Fort James Corporation (now owned by Georgia-Pacific).[7] In addition, EPA has identified Weyerhaeuser as an additional PRP. In 2000, Georgia-Pacific acquired Fort James (Georgia-Pacific, 2000). However, for consistency with previous documents, facilities operated by Fort James will continue to be discussed separately in this report.

Allied Paper, Inc. facilities include the former Monarch and Bryant Mills on Portage Creek in Kalamazoo and the former King Mill on Lake Street in Kalamazoo. Georgia-Pacific Corporation facilities include several mills on the bank of the Kalamazoo River along King Highway in Kalamazoo. Plainwell Inc. facilities include a mill on the bank of the Kalamazoo River along Allegan Street in Plainwell. The locations of the PRP facilities are shown in Figure 1.2.

6. Those operable units are OU1 – Allied Paper Property/Bryant Mill Pond Area, OU2 – Willow Boulevard and A-Site Landfill, OU3 – King Highway Landfill, OU4 – 12th Street Landfill, and OU5 – Portage Creek and Kalamazoo River sediments.

7. The term PRP as used in this document refers to parties potentially liable for cleanup costs or natural resource damages under CERCLA and/or under Section 20126a(1)(c) of Part 201, Environmental Remediation, of the Natural Resources and Environmental Protection Act (NREPA), MCL § 324.20126.

1.5 Spatial and Temporal Extent of the Stage I Assessment

The KRE Assessment Area (assessment area) includes the natural resources within the Portage Creek and Kalamazoo River riparian corridors that are exposed to hazardous substances released from the PRP facilities along with any area where hazardous substances released at or from the site have come to be located or where natural resources or the services they provide may have been affected by these hazardous substances.

Releases of PCBs into the KRE from paper company facilities began in the 1950s, as described in Chapter 2. However, the Stage I Assessment focuses on assessing injuries under current and recent (i.e., in the last two decades) conditions, since most of the available relevant data have been collected in the last two decades. Since current and recent PCB concentrations in the KRE are generally lower than in previous decades (Blasland, Bouck & Lee, 2000b), any current and recent PCB injuries have likely been occurring since the 1950s.

1.6 Organization of the Stage I Injury Assessment Report

Chapter 2 presents information on releases of hazardous substances and exposure pathways. Chapters 3 and 4 assess injuries to surface water and sediment, respectively, which together constitute the surface water resource. Chapter 5 describes fish consumption advisory injuries that result from PCB contamination in fish. Chapter 6 assesses other injuries to aquatic biological resources, and Chapter 7 assesses injuries to wildlife. Chapter 8 presents an assessment of indirect injuries to natural resources that can result from different remedial actions taken to address PCB contamination. Chapter 9 summarizes the conclusions of the Stage I Assessment. References cited in the text are provided at the end of the document.

2. PCB Releases and Pathways

This chapter summarizes information on the releases of PCBs from paper company facilities located along Portage Creek and the Kalamazoo River, and describes the transport pathways of the released PCBs in the KRE. The DOI regulations specify that a pathway determination be conducted as part of the injury determination phase of an NRDA [43 C.F.R. §11.63]. The purpose of the pathway determination is to determine the route through which the released hazardous substances are transported from the release points to the exposed natural resources [43 C.F.R. §11.61(c)(3)]. The pathway determination thus establishes the link between PCB releases into the KRE and the natural resources exposed to and injured by the PCBs. The presence of this link is important in establishing that the injuries in question result from the PCB releases from PRP facilities.

This chapter documents that the PRP facilities have released PCBs into the KRE and that the PRP facilities are the dominant source of PCBs to the KRE. Then the transport pathways of PCBs in the KRE are described. Finally, the PCB contamination that has resulted from PCB releases and transport in the KRE is described.

2.1 PCBs

PCBs constitute a class of chemical compounds that have differing numbers and positions of chlorine atoms substituted on a biphenyl ring (Figure 2.1) (Erickson, 1997). PCB formulations used in industrial processes, such as those released from KRE paper companies, were sold as commercial mixtures that contained many different individual PCB compounds (called "congeners"). PCBs were widely used in part because they are relatively resistant to chemical or biological degradation, and this property also makes them relatively persistent in the environment once released (Erickson, 1997). Monsanto Corporation, which was the sole commercial producer of PCBs in the United States, sold PCB mixtures under the trade name "Aroclor." Monsanto manufactured several Aroclor formulations that differed in the total amount of chlorine added to biphenyl (the last two numbers in most Aroclor designations indicate this percentage of chlorine, by weight, in the final mixture). Consequently, the mixture of congeners present in the formulation varies from Aroclors containing primarily lower chlorinated congeners (e.g., Aroclor 1242) to those containing primarily higher chlorinated congeners (e.g., Aroclor 1260; Erickson, 1997).

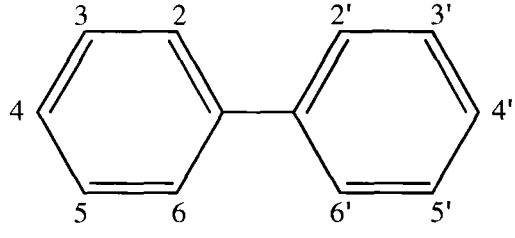

Figure 2.1. Biphenyl molecular structure.

There are 209 individual PCB congeners that differ in the number and position of chlorine atom substitutions on the biphenyl ring (Erickson, 1997). For example, there are three different monochloro biphenyl congeners that have the single chlorine atom attached to the biphenyl structure in different positions (positions 2, 3, and 4 in Figure 2.1 — positions 5 and 6 are equivalent to positions 3 and 2, respectively, when only a single chlorine atom is attached). The most highly chlorinated congener is decachloro biphenyl, which has chlorine atoms at each of the ten available positions shown in Figure 2.1.

Congeners vary in their physical, chemical, and toxicological properties in ways that are dependent on the number and position of the chlorine atoms. For example, congeners with fewer chlorine atoms tend to be more water soluble, have higher vapor pressures, and bioaccumulate in organisms less than congeners with more chlorine atoms (Erickson, 1997). Furthermore, congeners with chlorine atoms in the 4 or 4' position (in Figure 2.1) and without any chlorine atoms at the 2, 6, 2', or 6' positions, called the "coplanar" congeners because the two phenyl rings can lie flat relative to each other, tend to be the most toxic congeners to fish, birds, and mammals. The coplanar congeners cause toxicity to fish, birds, and mammals through the same toxicological mechanism as dioxin (or 2,3,7,8-tetrachloro-*p*-dibenzodioxin), and are sometimes referred to as "dioxin-like" PCB congeners. Different Aroclor mixtures differ in the absolute and relative amounts of the dioxin-like PCB congeners present, as well as in the amounts of the other congeners that can cause toxicity through other mechanisms. Therefore, it is the PCB congeners present in an Aroclor mixture that determine the environmental fate, transport, bioaccumulation, and toxicity of the PCBs once released into the environment.

2.2 PCB Releases from PRP Facilities

PCBs were released into the KRE from various paper company facilities in Kalamazoo and Plainwell (see Figure 1.2). In the process of deinking and repulping recycled paper, paper mills produced substantial quantities of PCB contaminated waste, which was released to the KRE [Kalamazoo River Study Group v. Rockwell Int'l Corp., 107 F. Supp. 2d 817 (W.D. Mich. 2000)]. When the recycled paper stream included carbonless copy paper with PCBs, PCBs were present in the paper mill waste streams. The Trustees have identified Allied Paper, Inc. and its parent company, Millennium Holdings, Inc. (Allied); the Georgia-Pacific Corporation (Georgia-Pacific); Plainwell Inc. (Simpson Plainwell); and the Fort James Operating Company, Inc. (Fort James) as PRPs for the PCB releases (see Section 1.4). The Trustees may consider notifying other PRPs at a later date as information becomes available.[1]

1. EPA has identified Weyerhaeuser as an additional PRP.

Figure 1.2 shows the general locations of PRP paper mills (or former paper mills) in the Kalamazoo and Plainwell areas. Allied mills include the former Monarch and Bryant Mills on Portage Creek in Kalamazoo and the King Mill on Lake Street in Kalamazoo. Georgia-Pacific mills include several mills on the bank of the Kalamazoo River in Kalamazoo. Simpson Plainwell mills include a mill on the bank of the Kalamazoo River in Plainwell. Fort James mills include the Paperboard Packaging Mill and the KVP Specialty Papers Mills in Kalamazoo. The facilities of these PRPs also include various landfills and other areas where PCB-containing paper waste was handled or disposed.

2.2.1 History of PCB use at PRP facilities

During the post-World War II period from 1946 to 1953, the National Cash Register Company (NCR) conducted research and development of carbonless copy paper using a micro-encapsulation technique for imprinting images (Appleton Papers, 1987). Commercial sales of carbonless copy paper, which became known as "NCR paper," began in March 1954 (Appleton Papers, 1987). Carbonless copy paper manufactured between 1954 and 1971 contained Aroclor 1242 (A1242) as an ink carrier or solvent. The A1242 was used as a solvent for certain dyes that were encapsulated in small spheres and applied to one or both sides of the paper during the coating process. The walls of the spheres would rupture and release the dye when subjected to pressure. The average A1242 content in a sheet of carbonless copy paper was 3.4% by weight (Carr et al., 1977; Rockwell Int'l Corp., 107 F. Supp. 2d 817).

Production records show that between 1957 and 1971, about 44,162,000 lbs of A1242 were used in the production of carbonless copy paper (Carr et al., 1977). This amount accounted for an estimated 28% of all the PCBs that the Monsanto Chemical Company (the sole domestic producer of PCBs) sold for plasticizer applications during this period, and 6.3% of Monsanto's total domestic sale of PCBs for the 15 year period from 1957-1971 (Carr et al., 1977; Rockwell Int'l Corp., 107 F. Supp. 2d 817). Information on production prior to 1957 is not available.

Aroclor 1254 (A1254), another commercial PCB mixture, was also used to a limited extent in printing inks beginning in 1968 (Rockwell Int'l Corp., 107 F. Supp. 2d 817). It is estimated that the total usage of A1254 in carbonless copy paper was 50,000 lbs (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

According to Carr et al. (1977), approximately 19% of carbonless copy paper was being recycled across the country in 1976 and a greater proportion may have been recycled in previous years. Assuming an average recycling effort of 20% for this paper over the 18-year period when PCBs were in the paper, then recycled paper streams across the country contained 20% of the 44 million lbs of PCBs used in the carbonless copy paper, a total of some 8.8 million lbs of PCBs in recycled paper pulp over the 18 years. Although PCB use in the manufacturing of carbonless

copy paper was discontinued in 1971, the paper recycled by the Kalamazoo area paper companies continued to contain PCBs for many years after 1971. For example, samples of waste paper that was recycled at a KRE Fort James mill in 1976 contained up to 11,313 mg/kg PCBs (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

The PCBs in the carbonless copy paper that paper mills deinked and repulped either became integrated into new paper products or became part of the paper company waste stream. Deinking and pulping the recycled stock would break the spheres in the paper containing the PCB-laden dyes. These PCBs were then distributed throughout the paper recycling process, including in the waste stream. Some of the PCBs in the carbonless copy paper, however, remained in the recycled pulp and subsequently were incorporated in the new paper products. For example, PCB concentrations up to 433 mg/kg were measured in paperboard used for cereal packaging in 1971 (Carr et al., 1977).

Deinking and repulping recycled paper produces a substantial quantity of paper waste. The handling and disposal of paper waste was similar at each of the PRP deinking mills from the mid-1950s to early 1970s. Figure 2.2 provides a schematic of the fate of paper waste from the mills. Raw paper waste, containing water, clay, and fibrous waste, was pumped to a clarifier that separated out much of the settleable solids. The waste from the clarifier included wastewater (effluent) and residual clay and fibrous solids (underflow, or residuals; Blasland, Bouck & Lee, 1992).

Typically, the effluent from the clarifier was either recycled through the process systems, discharged to the river, or discharged to a municipal wastewater treatment plant (WWTP) (Figure 2.2). The underflow from the clarifier was pumped into residuals dewatering lagoons (Figure 2.2) and allowed to dry by evaporation for several months. The resulting dried residuals, consisting mostly of the grey clay and wood fibers, were then removed from the dewatering lagoons and deposited in disposal areas (Blasland, Bouck & Lee, 1992). The disposal areas, which are discussed in detail in the following sections, were located along the banks of the Kalamazoo River near the mills.

Studies in the past 20 years have shown that much of the paper residuals from the late 1950s to the early 1970s contained highly elevated concentrations of PCBs as a result of the deinking and repulping of recycled carbonless copy paper.

Monitoring of direct PCB releases from PRP facilities into the Kalamazoo River and Portage Creek is limited because effluent or residuals were not tested for PCBs until several years after the use of PCBs in carbonless copy paper was discontinued in 1971. However, there is direct evidence of PCB contamination in drainages that lead from paper mills to surface water and in groundwater beneath the disposal areas of several paper company facilities. Additionally, PCB-

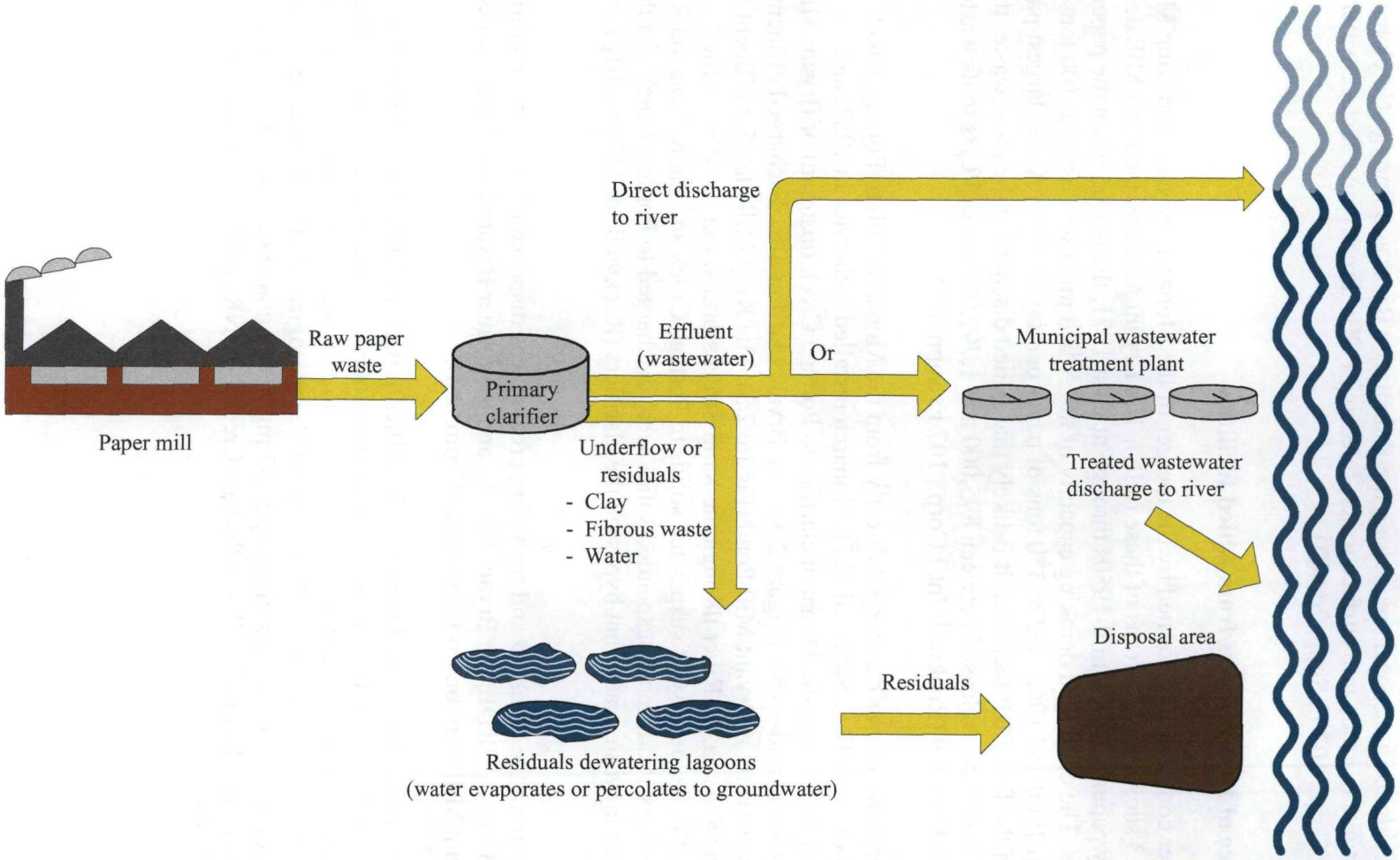

Figure 2.2. Typical fate of paper mill waste during the period when carbonless copy paper contained PCBs (1954-1971).

contaminated residuals in disposal areas along the Kalamazoo River and Portage Creek have eroded and continue to erode into surface water, releasing PCBs directly into surface water (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

2.2.2 Evidence of PCB releases from Allied facilities

The Allied Paper company was formed by the merger of the Monarch, Bryant, and King Mills in 1922 (Figure 2.3 shows the locations of these mills). The Bryant Mill and the King Mill deinked carbonless copy paper from the mid-1950s through at least 1971, the period when the paper contained PCBs. The combined deinking capacity of the Allied mills was listed at 100 tons of paper per day in 1960 and 1962, and at 350 tons of paper per day in 1965. As the largest paper manufacturer of the four PRP facilities, it is likely that Allied discharged the most waste. It has been estimated that Allied released between 895,000 and 1,790,000 lbs of PCBs in its waste stream from 1960 to 1979 (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

Before 1953, wastewater was discharged directly from the Monarch Mill to Portage Creek (Rockwell Int'l Corp., 107 F. Supp. 2d 817). Monarch installed a clarifier in 1953, and discharged the effluent from the Monarch clarifier to Portage Creek upstream of Bryant Mill Pond beginning in the mid-1950s (Figure 2.3). The Bryant clarifier also discharged effluent to Portage Creek upstream of Bryant Mill Pond (Figure 2.3). The King Mill clarifier effluent was discharged to the Kalamazoo River through the King Street storm sewer. In 1961, Allied discharged 156,494 lbs per day of suspended solids to Portage Creek and the Kalamazoo River through the clarifiers. Additional suspended solids were discharged to Portage Creek and the Kalamazoo River in waste water that bypassed the clarifiers (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

Residuals waste from the deinking and repulping process was disposed of at several facilities, including the Monarch HRDL, the Bryant HRDL, and the former Bryant Mill Pond, formed by the former Bryant Mill Dam on Portage Creek (Figure 2.3).

PCBs have been detected in several media on the Allied property (Table 2.1). The MDNR reported soil and sediment PCB concentrations in samples taken from a former channel that evidently discharged effluent or raw paper waste from the Bryant clarifier directly to the Bryant Mill Pond in Portage Creek (United Environmental Technologies, 1990). PCBs were detected in all 13 samples collected, at concentrations up to 37 mg/kg, demonstrating that the wastewater discharge in this channel carried PCBs to Portage Creek (MDNR, 1990b; United Environmental Technologies, 1990).

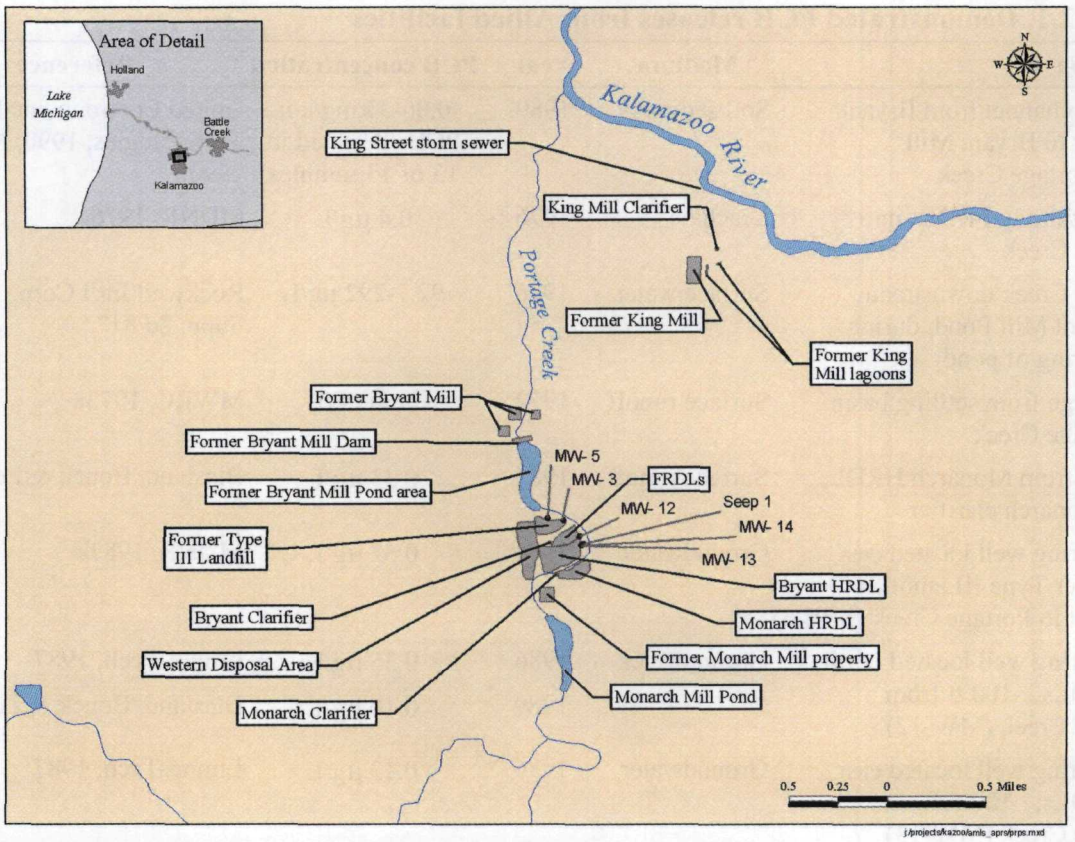

Figure 2.3. The Allied paper mills and surrounding facilities.

In 1976, the MDNR (1976a) analyzed leachate released from the former residuals decanting lagoons (FRDLs; still used at the time) into a ditch that fed directly to Portage Creek. PCBs were detected in the leachate at a concentration of 0.4 µg/L (Table 2.1; Figure 2.3).

In 1972, the Bryant Mill Pond was drained, washing sediments contaminated with PCBs downstream. In 1976, the water level in Bryant Mill Pond was again lowered, and sediments were washed downstream into the Kalamazoo River. Surface water samples collected in Portage Creek contained PCB concentrations ranging from 92.7 to 292 µg/L (Rockwell Int'l Corp., 107 F. Supp. 2d 817) (Table 2.1). Another sample of surface runoff draining from a settling basin to Portage Creek was collected by the Michigan Water Resources Commission (MWRC, 1973a) in 1972. The PCB concentration in this sample was reported at 56 µg/L (MWRC, 1973a). A sample collected in runoff from the Monarch HRDL in 1985 contained 0.33 µg/L PCBs (Blasland, Bouck & Lee, 1992).

Table 2.1. Demonstrated PCB releases from Allied facilities

Location	Medium	Year	PCB concentration	Reference
Former channel from Bryant clarifier to Bryant Mill Pond/Portage Creek	Soil/sediment	1989	0.06-37 mg/kg; PCBs detected in 13 of 13 samples	United Environmental Technologies, 1990; MDNR, 1990b
Ditch draining FRDLs into Portage Creek	Leachate	1976	0.4 µg/L	MDNR, 1976a
Portage Creek downstream of Bryant Mill Pond, during dewatering of pond	Surface water	1976	92.7-292 µg/L	Rockwell Int'l Corp., 107 F. Supp. 2d 817
Discharge from settling basin to Portage Creek	Surface runoff	1972	56.0 µg/L	MWRC, 1973a
Runoff from Monarch HRDL, near Monarch clarifier	Surface runoff	1985	0.33 µg/L	Blasland, Bouck & Lee, 1992
Monitoring well located east of former Type III landfill, adjacent to Portage Creek (MW-3)	Groundwater	1986	0.52 µg/L	MDNR, 1987a
Monitoring well located north of HRDLs, ~100 ft from Portage Creek (MW-12)	Groundwater	1986	0.35 µg/L	Limno-Tech, 1987
		1989	0.10 µg/L	Blasland, Bouck & Lee, 1992
Monitoring well located east of HRDLs, ~100 ft from Portage Creek (MW-13)	Groundwater	1986	0.13 µg/L	Limno-Tech, 1987
Monitoring well located east of HRDLs, ~20 ft from Portage Creek (MW-14)	Groundwater	1986	0.37 µg/L	Limno-Tech, 1987
Monitoring well located north of Bryant clarifier and former Type III Landfill, ~150 ft from Portage Creek (MW-5)	Groundwater	1985	2.1 µg/L 1.7 µg/L	Limno-Tech, 1987
		1986	1.4 µg/L	Blasland, Bouck & Lee, 1992
			0.56 µg/L	MDNR, 1987a
		1988	0.79 µg/L 0.61 µg/L 0.76 µg/L	Blasland, Bouck & Lee, 1992
		1989	3.3 µg/L	Blasland, Bouck & Lee, 1992

Table 2.1. Demonstrated PCB releases from Allied facilities (cont.)

Location	Medium	Year	PCB concentration	Reference
Seep located east of HRDLs, ~50 ft from Portage Creek (Seep 1)	Groundwater to surface water seep	1985-1986	26 µg/L[a] 10 µg/L[a] na/4.4 µg/L[b] 3.3/2.5 µg/L[b] 4.8/1.8 µg/L[b] 2.3/1.2 µg/L[b] 5.8/1.6 µg/L[b]	Blasland, Bouck & Lee, 1992

a. Samples reported to contain sediment particles resuspended during sampling.
b. Split samples. The value on the left is the PCB concentration reported by Allied; the value on the right is the PCB concentration reported by MDNR.

PCB contaminated groundwater has been found on the Allied property in several locations (Table 2.1). For example, groundwater PCB concentrations outside the former Type III landfill, adjacent to Portage Creek, were over 0.5 µg/L (MDNR, 1987a). The groundwater in three locations north of the Monarch and Bryant HRDLs and within 100 ft of Portage Creek contained 0.10 to 0.37 µg/L PCBs (Limno-Tech, 1987; Blasland, Bouck & Lee, 1992). One well north of the Bryant clarifier has consistently contained PCBs in groundwater at concentrations ranging from 0.56 to 3.3 µg/L (Limno-Tech, 1987; MDNR, 1987a; Blasland, Bouck & Lee, 1992).

PCBs were also detected at concentrations up to 26 µg/L in a groundwater seep near the bank of Portage Creek east of the Monarch and Bryant HRDLs (Table 2.1). In other samples from the same seep, PCBs ranged from 2.3 to 5.8 µg/L (Allied) or 1.2 to 4.4 µg/L (MDNR) in split samples (Blasland, Bouck & Lee, 1992). These data show that PCBs contaminated groundwater that is subsequently released at the surface through the seep.

PCB contamination has been found in the HRDLs at both the Monarch and the Bryant Mill sites (Table 2.2). The Monarch HRDL contains an estimated 170,000 yd^3 of waste and covers approximately 3 acres at an average depth of 15 feet (Blasland, Bouck & Lee, 1993, 1997).

Evidence gathered by Blasland, Bouck & Lee (1993) from samples collected before 1990 shows that the Monarch HRDL contained an average PCB concentration of 13 mg/kg, with a maximum concentration of 61 mg/kg. PCB concentrations in residuals samples collected in 1993 from the Monarch HRDL were up to 140 mg/kg. The Bryant HRDL originally contained an estimated 390,000 yd^3 of waste, covered approximately 13 acres, and was approximately 15 ft deep (Blasland, Bouck & Lee, 1993, 1997; Table 2.2). In 1999, approximately 150,000 yd^3 of dredged material from the former Bryant Mill Pond was disposed of in the Bryant HRDL and the FRDLs (Rockwell Int'l Corp., 107 F. Supp. 2d 817). In January 2000, the disposal area into which material from the former Bryant Mill Pond had been placed was stabilized with sheetpile

Table 2.2. PCB concentrations in residuals, sediment, and soil at the Allied Operating Unit

Location	Area (acres)[a]	Estimated volume (yd^3)	Approx. depth (ft)	PCB concentration pre-1990 (mg/kg)[a]		PCB concentration in native soil 1993 (mg/kg)[b]	PCB concentration in residuals 1993 (mg/kg)[b]
				Mean	Range		
Monarch HRDL	3	170,000[b]	15[a]	13	ND-61	ND-0.47	0.53-140
Bryant HRDL	13	390,000[b, c]	15[a]	47	ND-1,200	ND-100	3.0-650
FRDLs	5	62,000[b]	Up to 16[b]	—	—	ND-7.0	ND-19
Former Type III Landfill	7.8	250,000[a]	20[a]	ND	ND	ND-2.4	0.14-2,000
Area north of landfill	—	—	Up to 5[a]	4	0.012-37	—	—
Western Disposal Area	—	380,000[b]	Up to 20[b]	ND	ND	ND-0.41	ND-2,500
Former Bryant Mill Pond	22	150,000[c]	Up to 8[a]	84	ND-1,000	ND-510	14-60

ND = not detected.

a. Blasland, Bouck & Lee, 1993.

b. Blasland, Bouck & Lee, 1997.

c. These figures represent the status of the Bryant HRDL and the Bryant Mill Pond prior to remediation of the former Bryant Mill Pond Area. In September 1999, EPA removed 150,000 yd^3 of PCB contaminated sediments from the former Bryant Mill Pond area and placed them in the Bryant HRDL and the FRDLs (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

between the disposal area and Portage Creek (Blasland, Bouck & Lee, 2000b). Prior to the addition of sediments from the former Bryant Mill Pond in 1999, the Bryant HRDL contained an average PCB concentration of 47 mg/kg with a maximum of 1,200 mg/kg PCBs (Blasland, Bouck & Lee, 1993). For comparison, Portage Creek sediments upstream of the Monarch HRDL contained an average of 0.54 mg/kg PCBs, and the Michigan Part 201 generic residential cleanup criterion is 4.0 mg/kg.

The FRDLs cover 5 acres between the Bryant HRDL and the former Type III Landfill (Figure 2.3), and contain an estimated 62,000 yd^3 of waste (Blasland, Bouck & Lee, 1993, 1997). The former Type III Landfill covers an area of 7.8 acres and is estimated to contain 250,000 yd^3 of waste (Blasland, Bouck & Lee, 1993, 1997). PCB concentrations in residuals collected in 1993 from the FRDLs ranged from below detection to 19 mg/kg and in soil from the FRDLs ranged from below detection to 7.0 mg/kg. PCB concentrations measured in the former landfill were as high as 2,000 mg/kg (Blasland, Bouck & Lee, 1997). PCB concentrations in an area to the north of the landfill, bordering on Portage Creek, ranged from 0.012 to 37 mg/kg (Blasland, Bouck & Lee, 1993). In the early 1990s, erosion control measures were taken at the FRDLs and at the former Type III Landfill (Blasland, Bouck & Lee, 2000b).

The Western Disposal Area is estimated to contain 380,000 yd^3 of waste (Figure 2.3; Table 2.2) (Blasland, Bouck & Lee, 1997). PCB concentrations in residuals samples from this location are up to 2,500 mg/kg, and in soil samples are up to 0.41 mg/kg (Blasland, Bouck & Lee, 1997).

In September 1999, EPA removed 150,000 yd^3 of sediments containing approximately 10 tons (9,000 kg) of PCBs from the former Bryant Mill Pond (Rockwell Int'l Corp., 107 F. Supp. 2d 817; Blasland, Bouck & Lee, 2000b). Prior to this removal action, the mean and maximum measured PCB concentrations in samples in the Bryant Mill Pond were 84 mg/kg and 1,000 mg/kg, respectively (Table 2.2) (Blasland, Bouck & Lee, 1993, 1997). PCB concentrations in residuals samples collected in 1993 ranged from 14 to 60 mg/kg, and were up to 510 mg/kg in subsurface soil samples (Blasland, Bouck & Lee, 1997). Approximately 92% of verification samples collected after the removal action had PCB concentrations of less than 1 mg/kg (Blasland, Bouck & Lee, 2000b).

Little PCB sampling has been performed at the former King Mill site in Kalamazoo (Figure 2.3). The MDNR took three surface soil samples from the former King Mill property in 1987 (Creal, 1987). Two samples contained PCBs, at concentrations of 4.5 and 9.1 mg/kg. Blasland, Bouck & Lee (1994e) collected six cores from the former King Mill lagoons in 1994. PCB concentrations in these samples were up to 79 mg/kg (Blasland, Bouck & Lee, 1994e, 2000b). In 1999, approximately 11,000 yd^3 of residuals were removed from one of the former lagoons and disposed of at the King Highway Landfill (see Figure 2.4; Blasland, Bouck & Lee, 2000b). However, due to construction at the King Highway Landfill, excavation activities at the former King Mill lagoons stopped before all waste was removed (MDEQ, 1997b). During the 1950s and 1960s, the paper waste from the former King Mill lagoons was deposited at the A-Site Disposal Area (now owned by Georgia-Pacific). PCB concentrations in the A-Site Disposal Area are covered in the next section.

2.2.3 Evidence of PCB releases from Georgia-Pacific facilities

The Georgia-Pacific paper mills were owned and operated by the Kalamazoo Paper Company from 1899 to 1967. Originally the facility consisted of five mills, three for making paper products and two for finishing and converting (Figure 2.4). Mills 1 and 3 both performed deinking operations in the 1950s and 1960s. Mill 3 discontinued deinking in the late 1960s, was refurbished, and resumed operations in 1975. Mill 1 deinked continuously until the late 1970s (Blasland, Bouck & Lee, 1992). Georgia-Pacific deinked up to 200 tons of waste paper per day, second only to Allied in scale; it has been estimated that Georgia-Pacific released between 560,000 and 1,120,000 lbs of PCBs from its mills from 1960 to 1979 (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

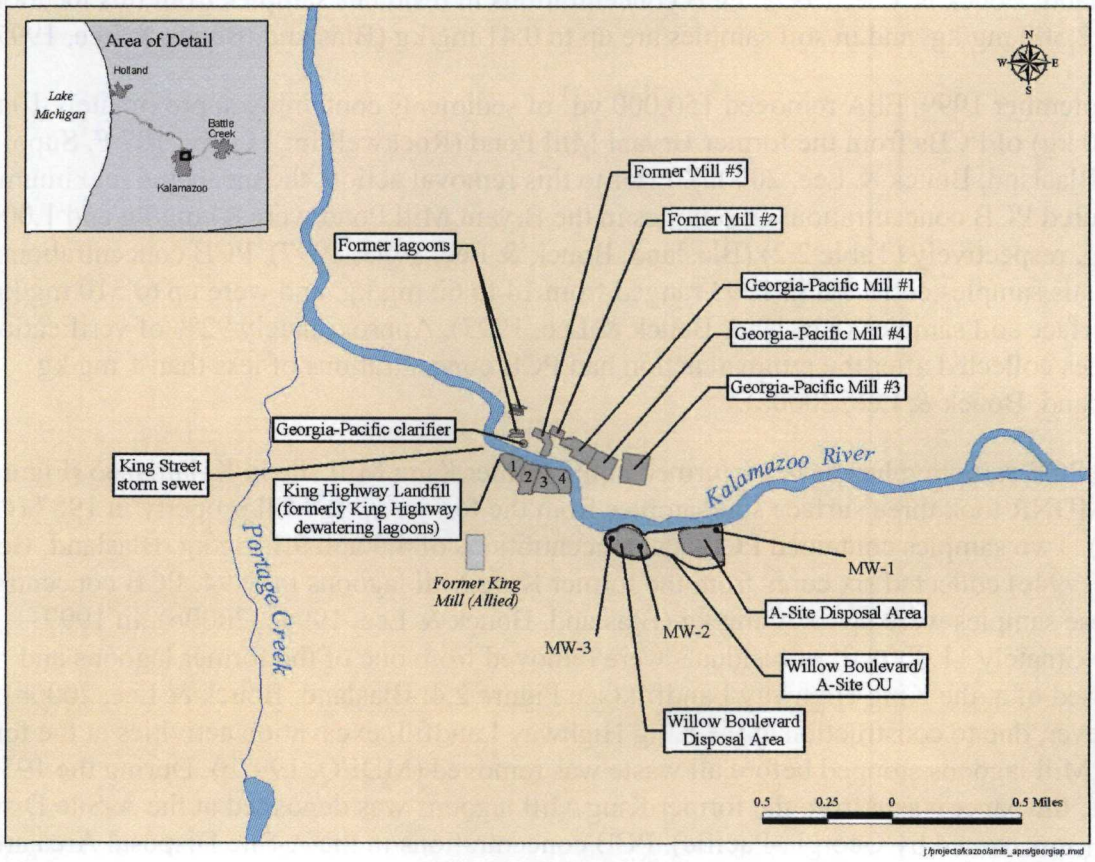

Figure 2.4. The Georgia-Pacific paper mill and surrounding facilities.

Before 1954, all industrial wastewater from Georgia-Pacific was discharged directly to the Kalamazoo River (Rockwell Int'l Corp., 107 F. Supp. 2d 817). Beginning in 1954, raw paper waste was routed to a clarifier. The clarifier effluent was pumped into the Kalamazoo River until 1964, when it was re-routed to the Kalamazoo WWTP. Figure 2.5 provides a flow chart of the fate of Georgia-Pacific paper waste from 1954 to the present.

The residuals from the Georgia-Pacific clarifier have been dewatered and disposed of at various locations (Figure 2.5). From 1954 until the late 1950s, the residuals were placed in the former lagoons next to the Georgia-Pacific clarifier. In the late 1950s, Georgia-Pacific began using the King Highway dewatering lagoons on the south side of the Kalamazoo River (site of the current King Highway Landfill; Figure 2.4) to dewater residuals. The original lagoons were then used as an emergency backup system when necessary.

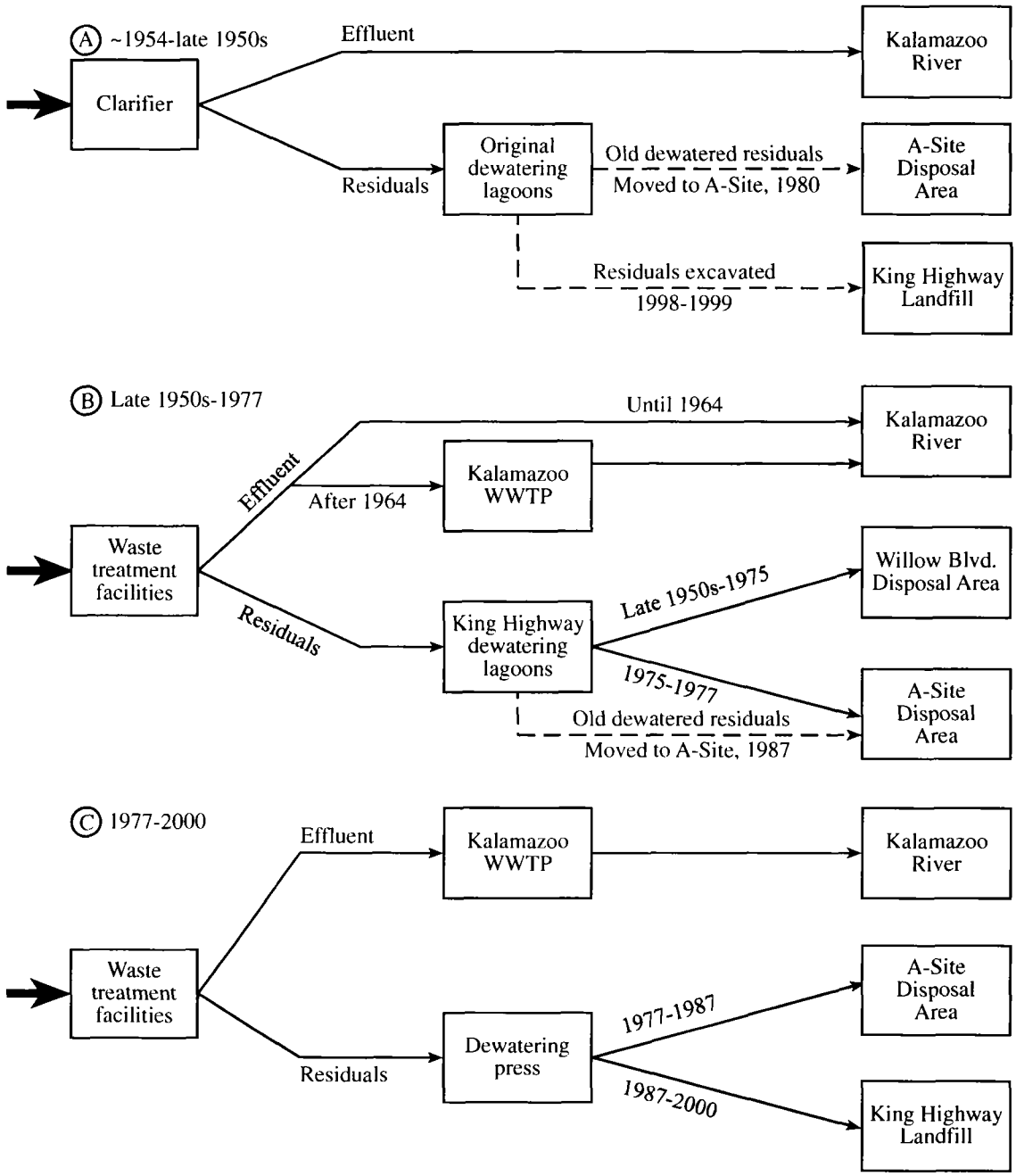

Figure 2.5. Simplified flow chart of the fate of Georgia-Pacific paper waste, 1954 to 2000.

Source: Blasland, Bouck & Lee, 1992.

From the late 1950s until 1977, Georgia-Pacific dewatered residuals in the King Highway dewatering lagoons. Until 1975, the dried residuals from the King Highway dewatering lagoons were excavated and disposed of at the Willow Boulevard Disposal Area (Figure 2.4). By 1975, the Willow Boulevard Disposal Area was filled to capacity, and Georgia-Pacific purchased the A-Site Disposal Area from Allied. Georgia-Pacific used the A-Site Disposal Area for residuals from 1975 until 1987 (Blasland, Bouck & Lee, 1992). In addition, residuals from the former lagoons next to the Georgia-Pacific clarifier were moved to the A-Site Disposal Area in 1980 (Blasland, Bouck & Lee, 1992).

In 1977, Georgia-Pacific began using dewatering presses that eliminated the need for dewatering lagoons (Blasland, Bouck & Lee, 1992). From 1977 until 1987, the residuals from the dewatering presses were deposited at the A-Site Disposal Area. In 1987, the King Highway dewatering lagoons were converted into a licensed Type III landfill. Since 1987, all Georgia-Pacific residuals have been deposited at the King Highway Landfill (Blasland, Bouck & Lee, 1992).

Cores were collected in the Georgia-Pacific former lagoons in 1994 (Figure 2.4). Despite the removal in 1980, residuals were found at a maximum depth of 4.5 ft (Blasland, Bouck & Lee, 1994e). PCBs were detected in both residuals and native soil samples collected from the former lagoons. Concentrations ranged from below detection (at a detection limit of 0.073 mg/kg) to 110 mg/kg in a surface sample. In 1998 and 1999, approximately 33,000 yd^3 of residuals were excavated from the former lagoons and 5,000 yd^3 of sediments were excavated from the floodplain adjacent to the lagoons. The materials were disposed of at the King Highway Landfill and riprap and geotextiles were used to stabilize soils (Blasland, Bouck & Lee, 2000b). After the removal of the residuals and verification sampling, the area of the former lagoons was backfilled, seeded, and stabilized with geotextile and riprap (MDEQ, 1997a).

Groundwater PCB contamination is evident in the areas near the A-Site and Willow Boulevard Disposal Areas. Table 2.3 shows elevated PCB concentrations in the groundwater beneath these two disposal areas. Elevated concentrations of PCBs have consistently been measured in the MW-1 well at the A-Site Disposal Area, located about 100 ft from the Kalamazoo River in the northeast corner of the disposal area. In 1988, concentrations measured at the well were up to 22 µg/L; however, it is possible that this elevated concentration was due to the disturbance from well drilling (Georgia-Pacific, 1988). Subsequent samplings have lower (though still elevated) concentrations of PCBs, ranging from 0.4 to 1 µg/L (Dell Engineering, 1989; Blasland, Bouck & Lee, 1992).

Table 2.3. PCB concentrations in groundwater beneath Georgia-Pacific disposal areas, 1988-1990

Location	Minimum PCB concentration (µg/L)	Mean PCB concentration (µg/L)	Maximum PCB concentration (µg/L)
A-Site Disposal Area (MW-1)	0.4	3.3	22.0
Willow Boulevard Disposal Area, west side (MW-3)	0.04	0.48	1.35
Willow Boulevard Disposal Area, east side (MW-2)	0.04	0.23	0.6

Sources: Dell Engineering, 1988, 1989; Georgia-Pacific, 1988; Blasland, Bouck & Lee, 1992.

In 1988, PCBs were detected in groundwater underlying the Willow Boulevard Disposal Area, approximately 100 to 150 ft from the Kalamazoo River. During one sampling event, the PCB concentration measured in well MW-3 on the west side of the disposal area was 1.35 µg/L, and the concentration measured at MW-2 on the east side of the disposal area was 0.6 µg/L (Dell Engineering, 1988) (Table 2.3). In two subsequent sampling events, the PCB concentrations at both wells were 0.06 µg/L during the first sampling event and 0.04 µg/L during the second sampling event (Dell Engineering, 1988).

PCBs have been released to the Kalamazoo River via the King Street storm sewer to the west of the King Highway Landfill. In 1976, the MDNR (1976c) analyzed a discharge from the storm sewer to the river and found 0.19 µg/L PCBs in the discharge. PCB concentrations in sediments in the storm sewer have been measured at 5.1 mg/kg in clay and 64 mg/kg in silt (Blasland, Bouck & Lee, 1992), and up to 190 mg/kg in sediments extending into the Kalamazoo River (Rockwell Int'l Corp., 107 F. Supp. 2d 817). It is not known whether the PCBs that have been discharged from this sewer originated at the former King Mill (Allied) or at the King Highway Landfill (Georgia-Pacific; S. Cornelius, MDEQ, personal communication). The volume of these residuals was estimated at over 33,000 yd^3 (Rockwell Int'l Corp., 107 F. Supp. 2d 817). In 1999, approximately 5,000 yd^3 of soils and residuals were excavated from the King Street storm sewer area and disposed of at the King Highway Landfill (Blasland, Bouck & Lee, 2000b). Riprap and geotextiles were used to stabilize the area along the river.

PCB contaminated waste has been found in all three of the Georgia-Pacific disposal areas (Willow Boulevard, A-Site, and King Highway Landfill; Table 2.4). The Willow Boulevard Disposal Area covers approximately 11 acres, on the south side of the Kalamazoo River (Figure 2.4), and contains 145,000 yd^3 of paper waste (Blasland, Bouck & Lee, 1995). PCB concentrations in surface residuals and soils collected in 1987 were up to 143 mg/kg, and up to 160 mg/kg in residuals and soils collected from the bottom layers of the disposal area (Swanson Environmental, 1987). PCB concentrations in residuals collected in 1993 ranged from below

Table 2.4. PCB concentrations in residuals/soils of Georgia-Pacific disposal areas

Location	Material	Year	PCB concentration (mg/kg)	Source
Willow Boulevard Disposal Area	Surface residuals/soil	1987	ND-143	Swanson Environmental, 1987
Willow Boulevard Disposal Area	Bottom residuals/soil	1987	3-160	Swanson Environmental, 1987
Willow Boulevard Disposal Area	Residuals	1993	ND-270	Blasland, Bouck & Lee, 1995
Willow Boulevard Disposal Area	Soil	1993	ND-4.7	Blasland, Bouck & Lee, 1995
Kalamazoo River adjacent to Willow Boulevard Disposal Area	Sediment/residuals	1993	0.16-44	Blasland, Bouck & Lee, 1995
A-Site Disposal Area	Bottom residuals/soils	1990	ND-14.8	Swanson Environmental, 1990
A-Site Disposal Area	Residuals	1993	ND-330	Blasland, Bouck & Lee, 1995
A-Site Disposal Area	Soil	1993	ND-61	Blasland, Bouck & Lee, 1995
King Highway Landfill — Cell 2	Residuals	1987	5.4	Blasland, Bouck & Lee, 1992
King Highway Landfill — Berm between Cell 1 and Kalamazoo River	Residuals	1989	ND-47.9	Environmental Resources Management, 1989
King Highway Landfill — Cell 2	Residuals two cores at approximately 13-30 in. deep	1991	ND-28	Blasland, Bouck & Lee, 1992; Environmental Resources Management, 1991
King Highway Landfill — Cells 1-4 and berm	Residuals/soil	1993	ND-310	Blasland, Bouck & Lee, 1994g

detection to 270 mg/kg, and in native soils from below detection to 4.7 mg/kg (Blasland, Bouck & Lee, 1995). In addition, the average PCB concentration in 5 sediment/residuals samples collected from the Kalamazoo River adjacent to the Willow Boulevard Disposal Area was 11 mg/kg (Blasland, Bouck & Lee, 1995). In 1999 and 2000, approximately 7,000 yd^3 of sediments were excavated and relocated within the Willow Boulevard Disposal Area (Blasland, Bouck & Lee, 2000b). A temporary sand cap was placed over the area at this time.

The A-Site Disposal Area is located to the east of the Willow Boulevard Disposal Area along the Kalamazoo River (Figure 2.4). It covers 23 acres, at depths of up to 27 ft, and is estimated to contain 475,400 yd^3 of paper waste (Blasland, Bouck & Lee, 1995). Residuals and soils collected from the bottom layers of the A-Site Disposal Area in 1990 contained PCB concentrations ranging from below detection to 14.8 mg/kg (Swanson Environmental, 1990). PCB

concentrations in residuals samples collected in 1993 ranged from below detection to 330 mg/kg, and in native soils from below detection to 61 mg/kg (Blasland, Bouck & Lee, 1995). In 1998, a 2,000 foot long sheetpile wall was installed along the edge of the A-Site Disposal Area to stabilize the berm that separates it from the Kalamazoo River (Blasland, Bouck & Lee, 2000b).

The King Highway Landfill is an operational licensed Type III landfill used for disposal of paper waste by Georgia-Pacific (Figure 2.4). The operable unit in the NPL site covers over 23 acres, 12.3 of which consist of cells 1-3 of the landfill (Blasland, Bouck & Lee, 1994g). Unit 4 covers 3.1 acres, was excavated to the water table before 1982, and collects surface water runoff. The remaining acreage contains access roads, dike slopes, berms, and buffer areas. The majority of the historical residuals from the former King Highway dewatering lagoons have been removed and placed in the Willow Boulevard or A-Site Disposal Areas. However, PCB concentrations indicate that not all PCB contaminated materials have been removed. A residuals sample collected from cell 2 in 1987 contained 5.4 mg/kg PCBs (Blasland, Bouck & Lee, 1992) and residuals collected from the berm between cell 1 and the Kalamazoo River in 1989 contained PCB concentrations as high as 47.9 mg/kg (Environmental Resources Management, 1989). Two cores collected from cell 2 contained concentrations up to 28 mg/kg (Environmental Resources Management, 1991; Blasland, Bouck & Lee, 1992). PCBs were detected in samples from all four cells and the berms collected in 1993 (Blasland, Bouck & Lee, 1994g). PCB concentrations in these samples were up to 310 mg/kg. Remedial actions begun in 1996 have been taken at the King Highway Landfill site, including relocation of residuals, the installation of a sheetpile wall, and construction of a Type III landfill cover system (Blasland, Bouck & Lee, 2000b). Georgia-Pacific ceased operations at the Kalamazoo plant in December 2000 (PaperAge, 2000; Georgia-Pacific, 2001).

2.2.4 Evidence of PCB releases from Simpson Plainwell facilities

The current Simpson Plainwell Mill was started in 1886 by the Michigan Paper Co., who operated the Plainwell Mill until 1956. Since then, the mill has been owned and operated by entities known as the Hamilton Paper Company (1956-1964), Weyerhaeuser (1964-1970), Plainwell Paper Company, Inc. (1970-1988), Simpson Plainwell Paper Co. (1988-1997), Plainwell Paper Company (1997-1998), and Plainwell, Inc. (1998-present) (Jackowski, 2002).

Carbonless copy paper was deinked at the Simpson Plainwell Mill from 1957 to 1962 (Blasland, Bouck & Lee, 1992; Figure 2.6). In 1962, the Simpson Plainwell Mill reported that 60 tons of paper per day was deinked (Rockwell Int'l Corp., 107 F. Supp. 2d 817). It has been estimated that Simpson Plainwell released between 254,000 and 507,000 lbs of PCBs from its mills from 1960 to 1979 (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

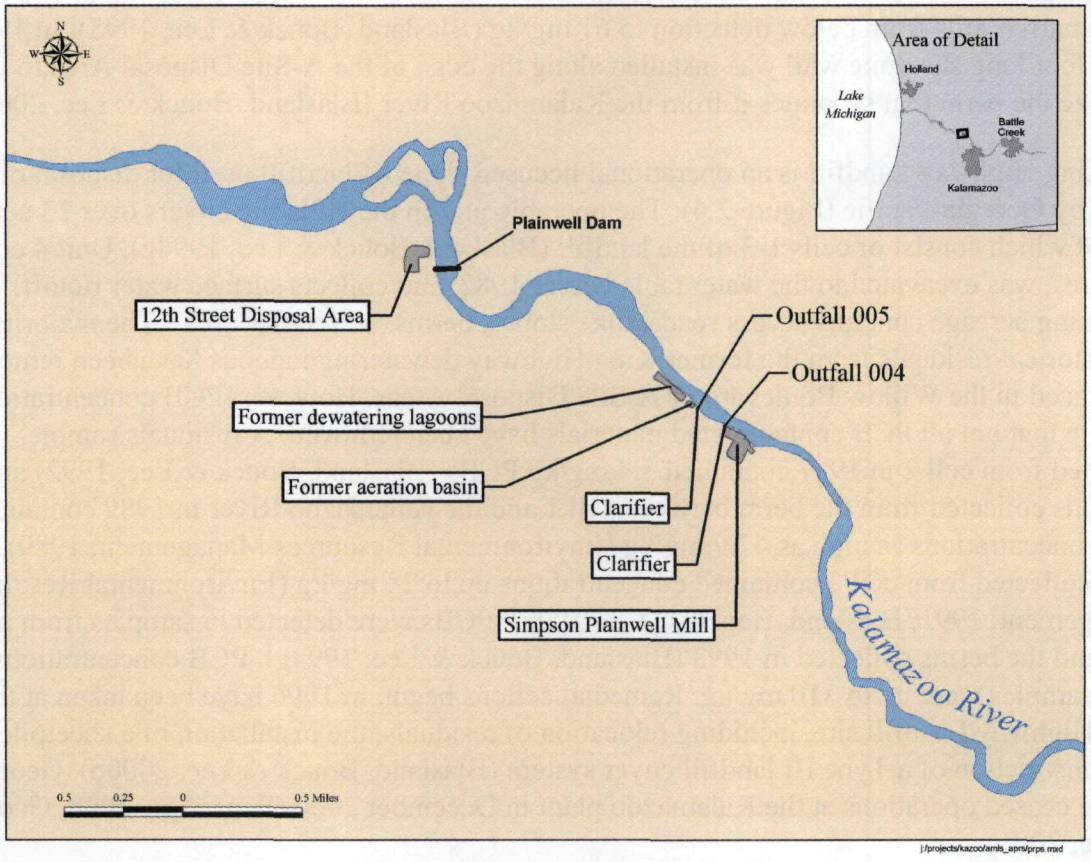

Figure 2.6. The Simpson Plainwell paper mill and surrounding facilities.

The treatment of the paper waste from the deinking operations was similar to that at the other Kalamazoo-area mills. Wastewater was discharged directly to the Kalamazoo River until 1954, when a clarifier was installed (Blasland, Bouck & Lee, 1994e; Rockwell Int'l Corp., 107 F. Supp. 2d 817). A document summarizing disposal of waste through 1960 indicates that 300 to 900 tons of paper were deinked per month and suspended solids discharges to the river averaged 14,000 to 34,000 lbs per day (Rockwell Int'l Corp., 107 F. Supp. 2d 817). After 1954, waste was pumped to the primary clarifier, clarifier effluent was pumped to the Kalamazoo River, and the underflow was pumped to on-site dewatering lagoons. The residuals from the lagoons were then trucked to the 12th Street Disposal Area for disposal (Figure 2.6). In 1981, the Plainwell Mill started using a new residuals dewatering process that supplanted the use of dewatering lagoons. At that time, the remaining residuals in the on-site lagoons were disposed of at the 12th Street Disposal Area, the lagoons were backfilled, and a new secondary clarifier and a treatment system were built on top of them (Blasland, Bouck & Lee, 1992).

PCBs have been detected in the Simpson Plainwell Mill effluent discharge. Table 2.5 summarizes data showing PCBs in effluent discharges; detectable PCB concentrations range from 0.039 to 0.13 µg/L. PCBs have not been detected in other effluent analyses from the Simpson Plainwell Mill.

Table 2.5. PCB concentrations in Simpson Plainwell Mill effluent

Effluent	Year	PCB concentration (µg/L)	Source
Clarifier effluent (Outfall 005)	1973	0.13	MWRC, 1975
Mill cooling water discharge (Outfall 004)	1978	0.10	MDNR, 1978b
Clarifier effluent (Outfall 005)	1978	0.10	
Unknown outfall	1985	0.039	Blasland, Bouck & Lee, 1992

Environmental PCB data related to the Simpson Plainwell Mill and 12th Street Disposal Area are limited. As of this writing, no PCB analyses of seep or runoff from the vicinity of the former dewatering lagoons on site or from the 12th Street Disposal Area have been published. One study conducted in 1994 did not find detectable PCBs in groundwater sampled from monitoring wells at the 12th Street Disposal Area at a detection limit of 0.00005 mg/L (Geraghty and Miller, 1994).

The 12th Street Disposal Area is located along the Kalamazoo River (Figure 2.6). No consistent berm or storm water collection system was used in this disposal area, and the current berm is constructed of paper residuals mixed with sand and gravel (Rockwell Int'l Corp., 107 F. Supp. 2d 817). Residuals in the disposal area have been measured to be as thick as 24.5 ft, and the total volume of waste in the landfill is approximately 200,000 yd^3 (Geraghty and Miller, 1994). Residuals are largely within the disposal area, but extend as far as 60 ft beyond the current berm of the disposal area (Geraghty and Miller, 1994). Several investigations have found elevated soil PCB concentrations in and near the disposal area (Table 2.6). Split samples of residuals were collected by MDNR and the Plainwell Paper Company in June 1987. MDNR recorded PCB concentrations up to 39 mg/kg, and the Institute of Paper Chemistry (for the Plainwell Paper Company) found 18 mg/kg PCBs in the split of that sample (RMT Engineering and Environmental Management Services, 1990; Blasland, Bouck & Lee, 1992). Ten of fourteen sludge and soil samples collected by the Plainwell Paper Company in September 1987 contained elevated PCBs, with a maximum recorded concentration of 21 mg/kg (RMT Engineering and Environmental Management Services, 1990; Blasland, Bouck & Lee, 1992). Similarly, all six samples collected in January 1989 contained elevated PCBs, including one sample with a concentration of 120 mg/kg (RMT Engineering and Environmental Management Services, 1990;

Table 2.6. PCB concentrations in residuals and soil samples collected at the Simpson Plainwell Mill and the 12th Street Disposal Area, 1987-1994

Location	Date	Number of samples (detected/total)	Minimum PCB concentration (mg/kg)	Mean PCB concentration (mg/kg)	Maximum PCB concentration (mg/kg)
12th St. Disposal Area, outside berm	June 1987[a]	3/3	2.6 (5)[b]	23.9 (12.3)[b]	39 (18)[b]
12th St. Disposal Area, outside berm	Sept. 1987[a]	10/14	< 0.5	7.4[c]	21
12th St. Disposal Area, outside berm	Jan. 1989[a]	6/6	0.16	30.0	120
12th St. Disposal Area, test pits	May 1993[d]	63/83	< 0.035	15.53[c]	158
Simpson Plainwell Mill, former aeration basin and dewatering lagoons	June 1994[e]	13/18	< 0.05	0.25[c]	1.6
Simpson Plainwell Mill, near mill	June 1994[e]	9/12	< 0.04	20.7[c]	240

a. Sources: RMT Engineering and Environmental Management Services, 1990; Blasland, Bouck & Lee, 1992.
b. Split sample. The first value is the result presented by the MDNR. Samples analyzed by the Institute of Paper Chemistry on behalf of Plainwell Paper are presented in parentheses.
c. A value of ½ the detection limit was used to calculate the mean in samples where PCBs were not detected.
d. Source: Geraghty and Miller, 1994.
e. Source: Blasland, Bouck & Lee, 1994e.

Blasland, Bouck & Lee, 1992). Finally, 63 of 83 samples collected from test pits throughout the 12th Street Disposal Area in 1993 contained detectable PCB concentrations. The mean PCB concentration in these samples was 15.53 mg/kg, and the maximum concentration was 158 mg/kg (Geraghty and Miller, 1994). Remedial activities conducted in 1984 included the capping of the disposal area (Blasland, Bouck & Lee, 2000b).

Additional sampling was conducted by Blasland, Bouck & Lee (1994e) in 1994 near the Simpson Plainwell Mill building and in the former aeration basin and dewatering lagoons area (Figure 2.6). PCBs were detected in samples from both locations (Table 2.6). Concentrations in the former aeration basin and dewatering lagoons averaged 0.25 mg/kg PCB with a maximum concentration of 1.6 mg/kg. Samples collected near the mill building had an average concentration of 20.7 mg/kg. The maximum concentration, 240 mg/kg PCB, was in a sample collected from the end of a discharge pipe that historically conveyed water to the Kalamazoo

River (Blasland, Bouck & Lee, 1994e). Additionally, sediment sampled from a sump along an outfall on the Simpson Plainwell Mill property in 1996 contained PCBs at a concentration of 7.2 mg/kg (Blasland, Bouck & Lee, 2000b). Drainage pipes leading to that outfall were cleaned in 1997 and sediment and flush water were disposed of off-site.

2.2.5 Evidence of PCB releases from Fort James facilities

The Fort James Corporation and its predecessors (James River, KVP Sutherland, and Brown Company) have operated two mills along the Kalamazoo River since 1939 (Rockwell Int'l Corp., 107 F. Supp. 2d 817). The first, the Fort James KVP Mill (consisting of two mill buildings), is located in Parchment, and the second, the Fort James Paperboard Mill (formerly known as the Sutherland Mill), is located in Kalamazoo (Figure 2.7). From 1939 to the mid-1970s wastewater from lagoons at the KVP Mill was discharged directly to the Kalamazoo River. A clarifier and sludge dewatering system was installed in the mid- to late 1970s. At the Paperboard Mill, wastewater was discharged to the Kalamazoo River until the late 1960s. It has been estimated that between 512,000 and 1,025,000 lbs of PCBs were released from the Fort James mills from 1960 to 1979 (Rockwell Int'l Corp., 107 F. Supp. 2d 817).

The Fort James Disposal Area covers approximately 40 acres to the east of the Kalamazoo River (Wilkins & Wheaton Testing Laboratory and STS Consultants, 1986). A Type II landfill of approximately 15 acres was used for disposal of general mill waste as early as the 1930s (Figure 2.7; STS Consultants, 1989). Between the 1960s and 1981, when the former Type II landfill was closed, residuals were the primary material disposed of at the landfill. Another Paper Residuals Disposal Area of approximately 25 acres, to the north of the disposal area, received residuals as well (Figure 2.7). Both the KVP Mill and the Paperboard Mill disposed of paper waste residuals at the Fort James Disposal Area (Wilkins & Wheaton Testing Laboratory and STS Consultants, 1986).

Samples of waste paper to be recycled in the Paperboard Mill collected in 1976 contained PCBs with reported concentrations up to 11,313 mg/kg (Rockwell Int'l Corp., 107 F. Supp. 2d 817). In the same year, samples of filtered solid waste from the mill had reported PCB concentrations of between 12.7 and 125.7 mg/kg (Rockwell Int'l Corp., 107 F. Supp. 2d 817). Another solid waste sample collected in 1977 had a PCB concentration of 180.6 mg/kg.

PCBs have been detected in discharge from the Fort James mills (Table 2.7). Concentrations as high as 0.65 µg/L have been detected in samples collected in 1972 of cooling and waste water discharged to the Kalamazoo River (MWRC, 1972b). Additionally, a PCB concentration of 0.04 µg/L was detected in one groundwater sample collected from the Fort James Disposal Area in 1988 (STS Consultants, 1989).

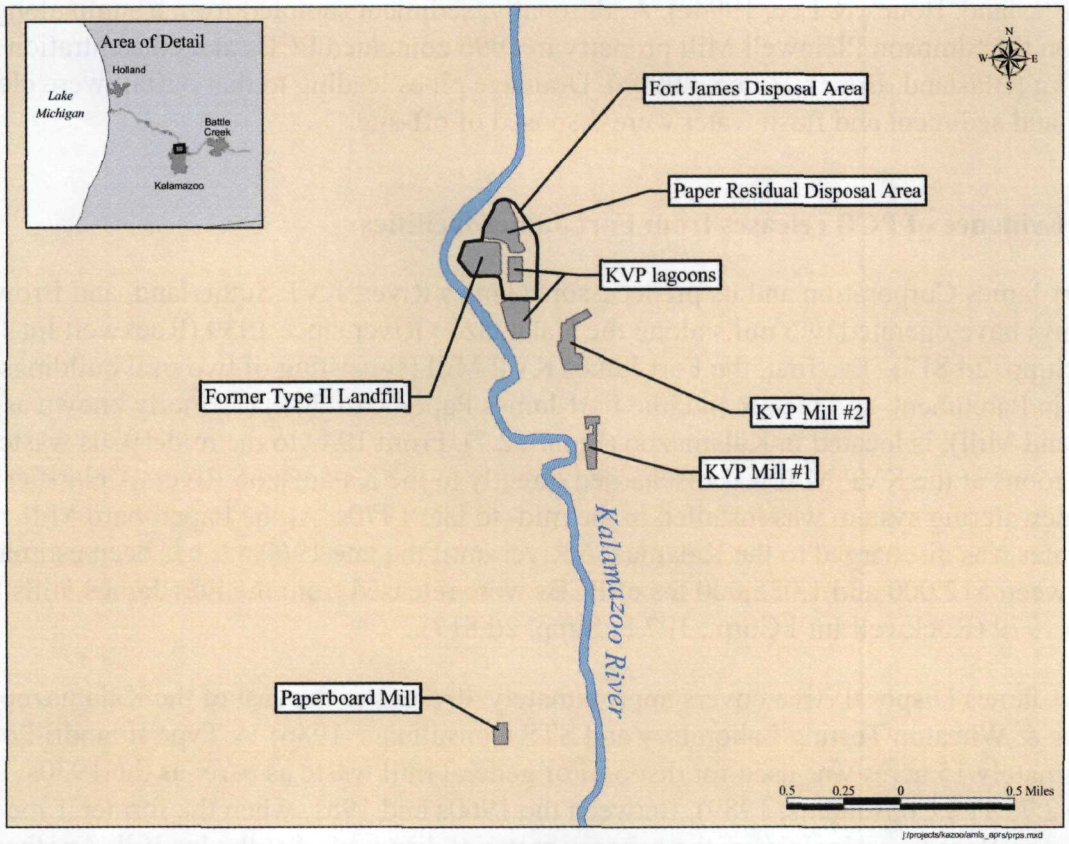

Figure 2.7. The Fort James mills and surrounding facilities. See Figure 1.2 for general location.

Table 2.7. PCBs detected in discharge from Fort James mills

Location	Sample date	PCB concentration (µg/L)	Source
KVP Mill #1 discharge to Kalamazoo River	August 22, 1972 August 23, 1972	0.65 0.39	MWRC, 1972b
KVP Mill #2 discharge to Kalamazoo River	April 3, 1973 August 16, 1976	0.31 0.17	MWRC, 1973b; MDNR, 1976b
KVP Lagoon discharge to Kalamazoo River	April 3, 1973 May 19, 1987	0.16 0.11	MWRC, 1973b; Koivuniemi, 1987

Soil and residuals samples collected from locations in the Fort James Disposal Area in 1987 (see Figure 2.7) contained PCBs. PCB concentrations in these samples as reported by MDNR ranged from below detection (at a detection limit of 0.75 mg/kg) to 7.3 mg/kg (Koivuniemi, 1987). Split samples from these same locations were also analyzed by laboratories on behalf of Fort James, and reported results ranged from below detection (at a detection limit of 0.1 mg/kg PCB) to 2.2 mg/kg PCB (James River Corporation, 1988). Additional samples from the Fort James Disposal Area analyzed on behalf of Fort James ranged from less than 0.01 mg/kg PCB to 1.81 mg/kg PCB (James River Corporation, 1988).

2.3 PCB Transport Pathways

Once PCBs are released into Portage Creek or the Kalamazoo River, the primary transport pathway is downstream movement with the flowing surface water (Blasland, Bouck & Lee, 2000b). The PCBs can be dissolved in the water or attached to particulate matter (e.g., sediment, plant material) as it moves downstream. As the PCBs adsorbed to particulate matter move downstream, they can be deposited in bed, bank, or floodplain areas of the river. These PCBs can be buried permanently in place by the deposition of additional sediment, or buried temporarily if erosion or scouring causes the PCBs to be remobilized and transported farther downstream.

Blasland, Bouck & Lee (2000b) calculated annual mass loads of PCBs in Portage Creek and at several locations along the Kalamazoo River using surface water data collected in 1994. The annual PCB load contributed by Portage Creek into the Kalamazoo River was estimated at 4.2 kg (Table 2.8). Annual PCB loads in the Kalamazoo River increase from an estimated 10 kg/year at River Street in Kalamazoo to an estimated 28 kg/year in Plainwell. The estimated load at the M-89 bridge downstream of Allegan Dam is 25 kg/year.

Table 2.8. Estimated annual PCB load in Portage Creek and locations along the Kalamazoo River, 1994

Location	Estimated PCB load[a] (kg/year)
Portage Creek	4.2
Kalamazoo River	
River Street, Kalamazoo	10
Michigan Avenue, Kalamazoo	12
Farmer Street, Plainwell	28
M-222, Allegan	26
M-89, downstream of Allegan Dam	25

a. Loads were estimated using a flow-stratified method that takes into account different PCB concentrations at different flow rates.
Source: Blasland, Bouck & Lee, 2000b.

The MDEQ conducted a study on PCB concentrations and loadings across Morrow Pond and Lake Allegan using monthly data collected from May 2001 — October 2002 (Camp Dresser & McKee, 2003a). Loadings of PCBs into and out of Morrow Pond ranged from 0.001 lbs/day to 0.025 lbs/day. The average loading both into and out of Morrow Pond over the period of study was 0.009 lbs/day. Thus MDEQ concluded that Morrow Pond was neither a source, nor a sink for PCBs. Loading at the inlet of Lake Allegan ranged from 0.014 lbs/day to 0.199 lbs/day of PCBs with an average of 0.1 lbs/day, while loading at the outlet was much lower, ranging from 0.001 lbs/day to 0.048 lbs/day and an average 0.015 lbs/day. These data suggest that Lake Allegan was acting as a sink for PCBs from upstream areas in the Kalamazoo River, as well as acting as an ongoing source for downstream areas.

There is also evidence that PCBs are transported by the Kalamazoo River into Lake Michigan via the surface water pathway. Data from a 1994-1995 mass balance study on PCB loadings show that the Kalamazoo River contributed approximately 36.8 kg (or 12%) of the 304.4 kg of PCBs contributed annually to Lake Michigan by its tributaries during this time period (Figure 2.8; U.S. EPA, 2000). From 1994-1995, the Kalamazoo River was the third largest tributary contributor of PCBs to the lake, behind the Lower Fox River (186 kg/yr) and the Grand Calumet River (37.2 kg/yr), and the largest contributor on the eastern shore of Lake Michigan (Figure 2.9; U.S. EPA, 2000). Therefore, these data demonstrate that PCBs are transported from the Kalamazoo River into Lake Michigan.

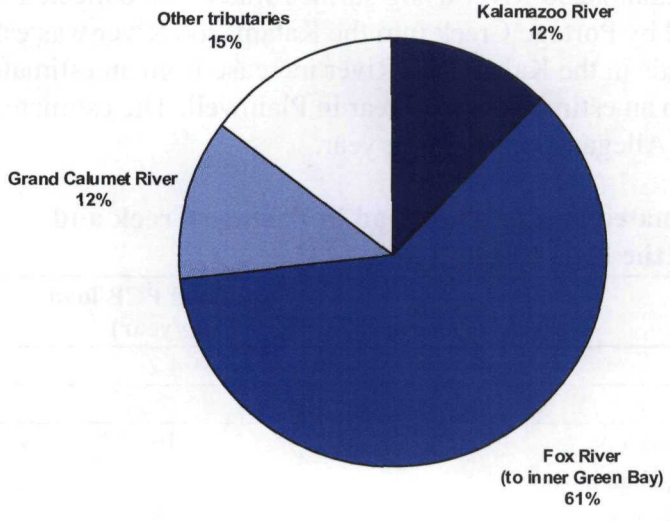

Figure 2.8. 1994-1995 tributary loadings of PCBs to Lake Michigan.

Source: U.S. EPA, 2000.

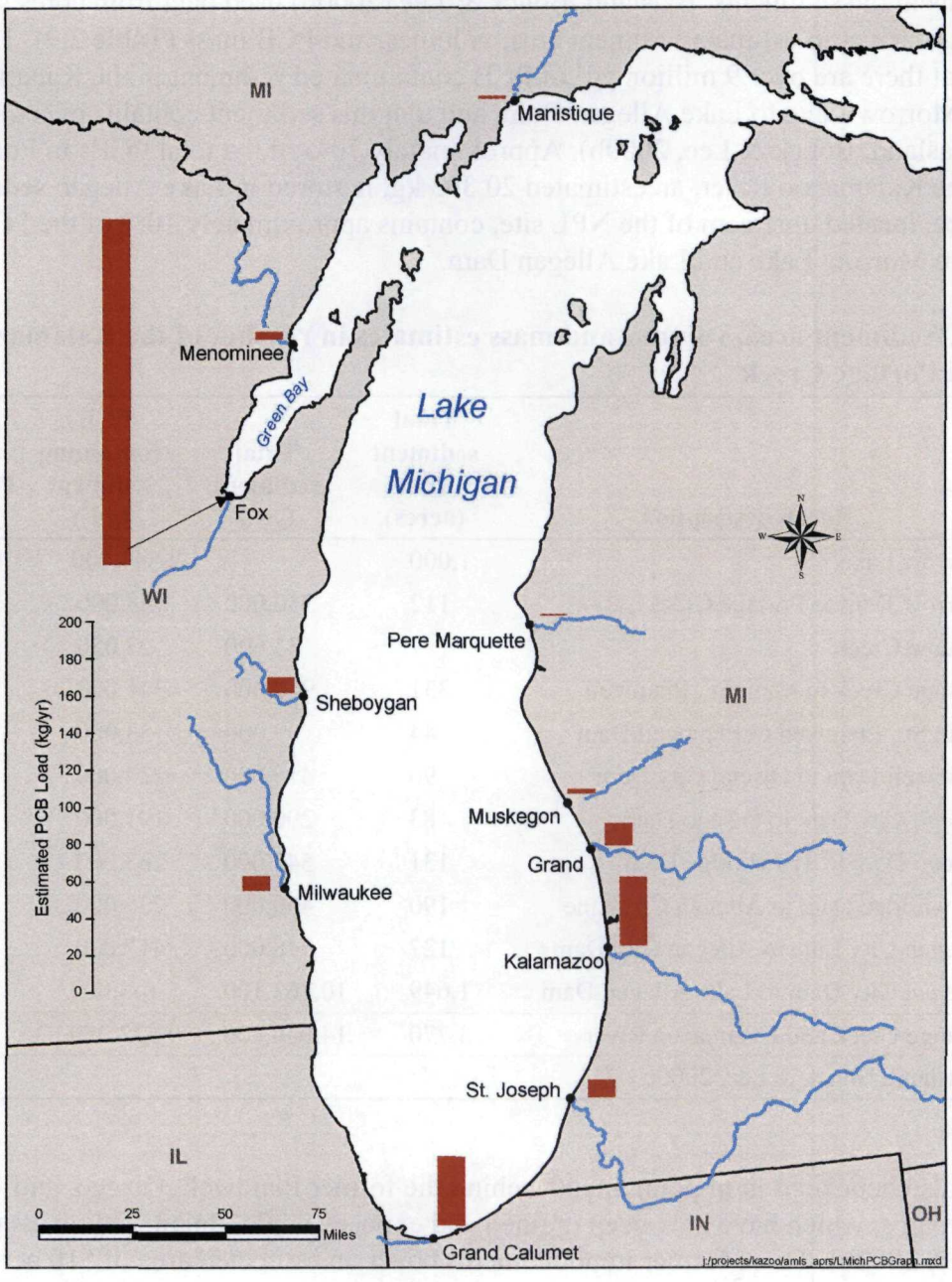

Figure 2.9. Spatial distribution of 1994-1995 tributary loadings of PCBs to Lake Michigan.

PCBs were deposited in instream sediments of the Kalamazoo River, and a large quantity of PCBs remains in the sediments. Blasland, Bouck & Lee (2000b) used data from cores sampled in the Kalamazoo River to estimate sediment area, volumes, and PCB mass (Table 2.9). They estimated that there are over 9 million yd^3 of PCB contaminated sediment in the Kalamazoo River from Morrow Lake to Lake Allegan Dam, and that this sediment contains over 29,000 kg of PCBs (Blasland, Bouck & Lee, 2000b). Approximately 70% of the total PCBs in Portage Creek and the Kalamazoo River, an estimated 20,363 kg, is stored in Lake Allegan sediments. Morrow Lake, located upstream of the NPL site, contains approximately 10% of the PCBs in the river between Morrow Lake and Lake Allegan Dam.

Table 2.9. Sediment area, volume, and mass estimates in reaches of the Kalamazoo River and Portage Creek

Reach	Reach description	Total sediment area (acres)	Total sediment (yd^3)	PCB-containing sediment (yd^3)	PCB mass (kg)
—	Morrow Lake	1,000	—	2,541,000	2,831
A1	Morrow Dam to Portage Creek	112	350,000	58,000	345
—	Portage Creek	7	33,600	23,050	162
A2	Portage Creek to Main St., Plainwell	331	950,000	341,000	754
B	Main St., Plainwell to Plainwell Dam	44	99,000	53,000	241
C	Plainwell Dam to Otsego City Dam	96	415,000	224,000	695
D	Otsego City Dam to Otsego Dam	83	290,000	191,000	306
E	Otsego Dam to Trowbridge Dam	131	542,000	263,000	719
F	Trowbridge Dam to Allegan City Line	190	450,000	258,000	476
G	Allegan City Line to Allegan City Dam	127	748,000	417,000	2,562
H	Allegan City Dam to Lake Allegan Dam	1,649	10,163,100	5,143,000	20,363
Total	Portage Creek and Kalamazoo River	3,770	14,040,700	9,512,050	29,484

Source: Blasland, Bouck & Lee, 2000b.

PCBs were also deposited in impoundments behind the former Plainwell, Otsego, and Trowbridge dams, which have now been drained and exposed as floodplain sediments (Blasland, Bouck & Lee, 2000b). These former impoundments cover an estimated area of 510 acres, and contain close to 3 million yd^3 of sediment (Table 2.10). Estimates of PCB mass made using cores collected by Blasland, Bouck & Lee (2000b) suggest that there are approximately 3,200 kg of PCBs in the former Plainwell impoundment, 6,300 kg of PCBs in the former Otsego impoundment, and 15,400 kg of PCBs in the former Trowbridge impoundment (Table 2.10).

Table 2.10. Former impoundment size and PCB mass estimates

Former impoundment	Area (acres)	Average depth (ft)	Volume (yd^3)	PCB mass (kg)
Plainwell	59	3.8	360,000	3,200
Otsego	77	4.4	540,000	6,300
Trowbridge	374	3.1	1,900,000	15,400

Source: Blasland, Bouck & Lee, 2000b.

PCBs are continuing to be released into the Kalamazoo River. The sources of these releases include erosion from PCB contaminated soils along the banks above the Plainwell, Otsego and Trowbridge Dams; re-releases from river sediment; periodic flooding of these soils; surface water runoff from PCB contaminated residuals at the Willow Boulevard and A-Site Disposal Areas; and groundwater containing PCBs that reaches the ground surface near the Bryant Mill Pond and flows into Portage Creek.

2.4 PCB Contamination and Persistence in the KRE

2.4.1 PCB contamination in the KRE

PCB contamination in the KRE has been documented by many different investigators over the years. Because of their persistence and tendency to bioaccumulate, PCBs can contaminate entire ecosystems once they are introduced into the environment. Such is the case in the KRE.

Studies have documented the presence of PCB concentrations greater than reference area concentrations in KRE abiotic media, such as surface water, sediment, and soil. Figures 2.10-2.12 show the distribution of PCBs in surface sediment and surface floodplain soils throughout the KRE. Concentrations are low in Morrow Lake, increase downstream of PRP facilities, and remain elevated throughout the KRE downstream to Lake Michigan. The highest PCB concentrations are seen in depositional areas of the river, behind current and former dams.

Similarly, PCB concentrations greater than reference area concentrations have been documented in many different kinds of biota, including many species of plants, invertebrates, fish, amphibians, reptiles, birds, and mammals (Figure 2.13). PCBs are present in every component of the KRE ecosystem that has been studied to date, including in the entire aquatic, terrestrial, and wetlands-based food chains.

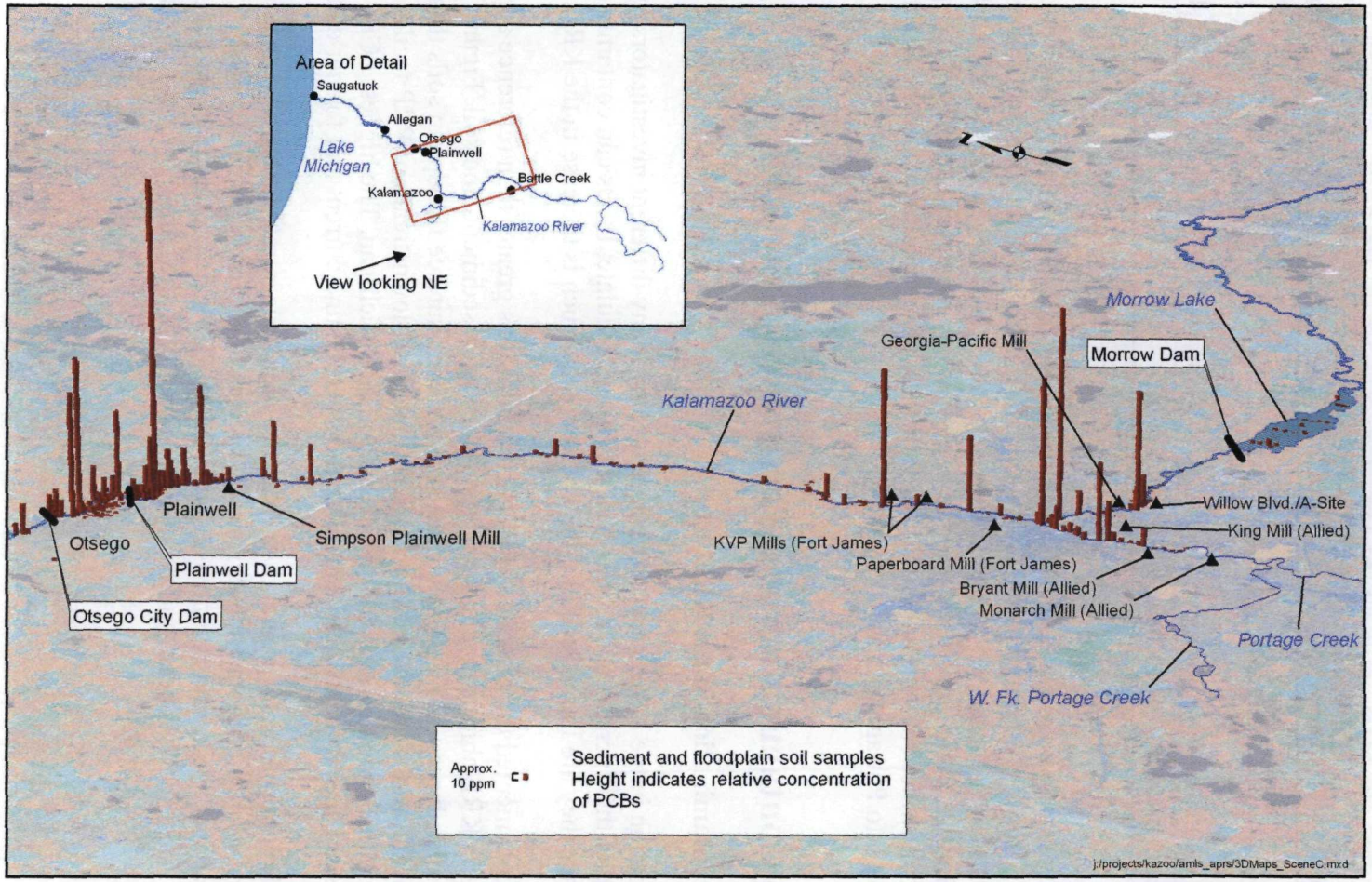

Figure 2.10. PCB concentrations in surface sediments and floodplain soils from Morrow Lake to the Otsego City Dam.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a.

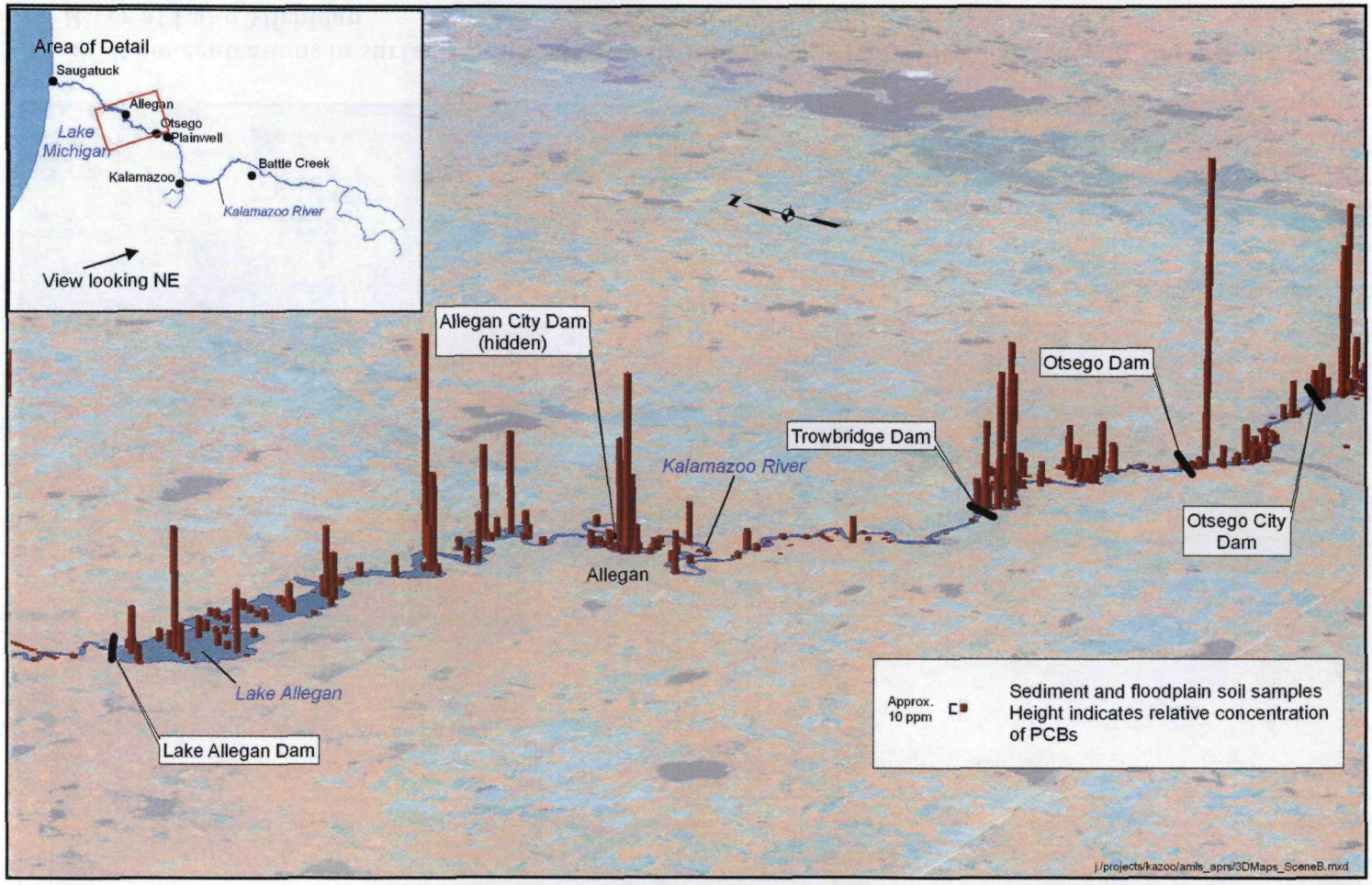

Figure 2.11 PCB concentrations in surface sediments and floodplain soils from the Otsego City Dam to the Lake Allegan Dam.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a.

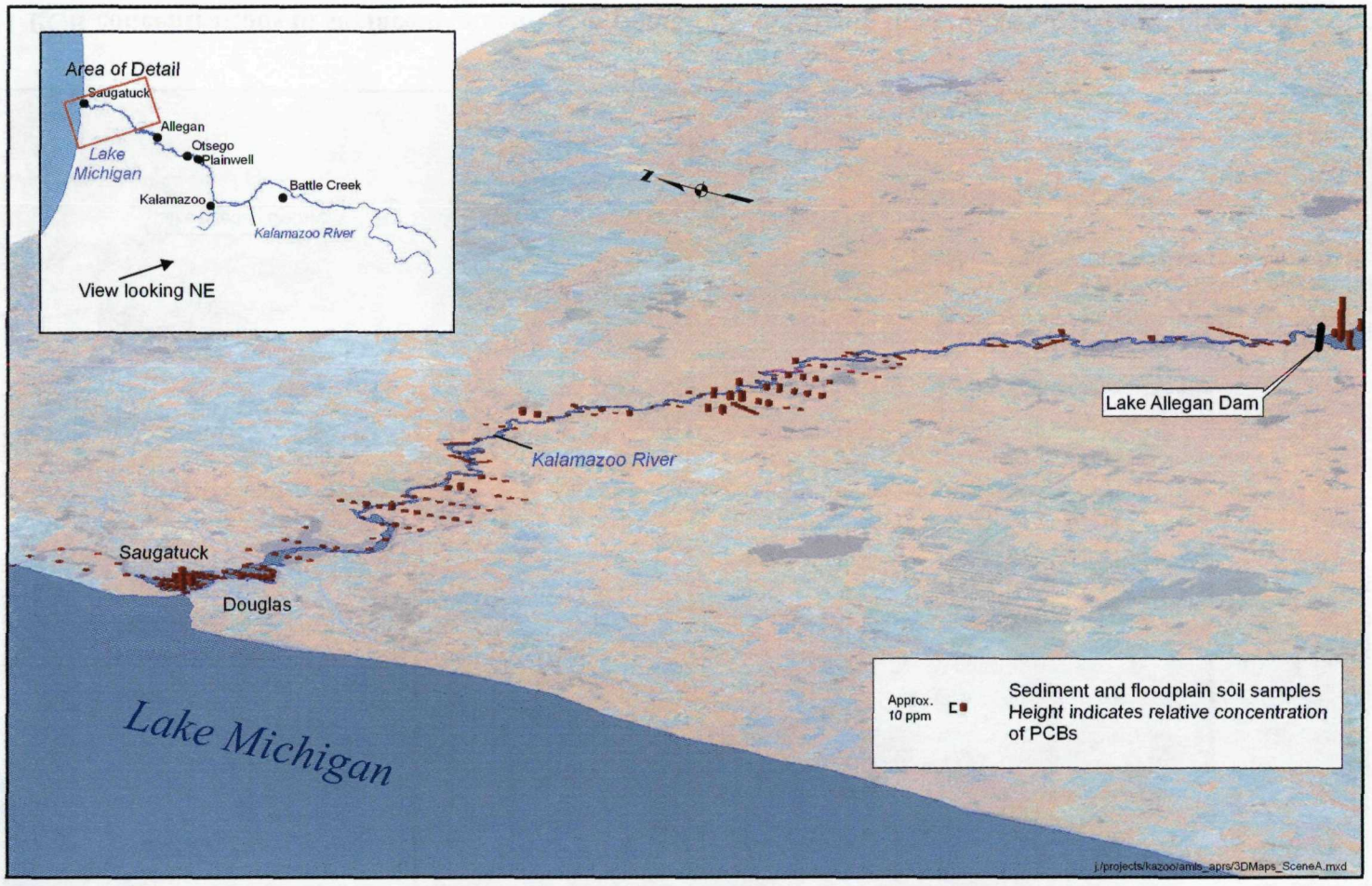

Figure 2.12. PCB concentrations in surface sediments and floodplain soils from the Lake Allegan Dam to the mouth of the Kalamazoo River at Lake Michigan.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a.

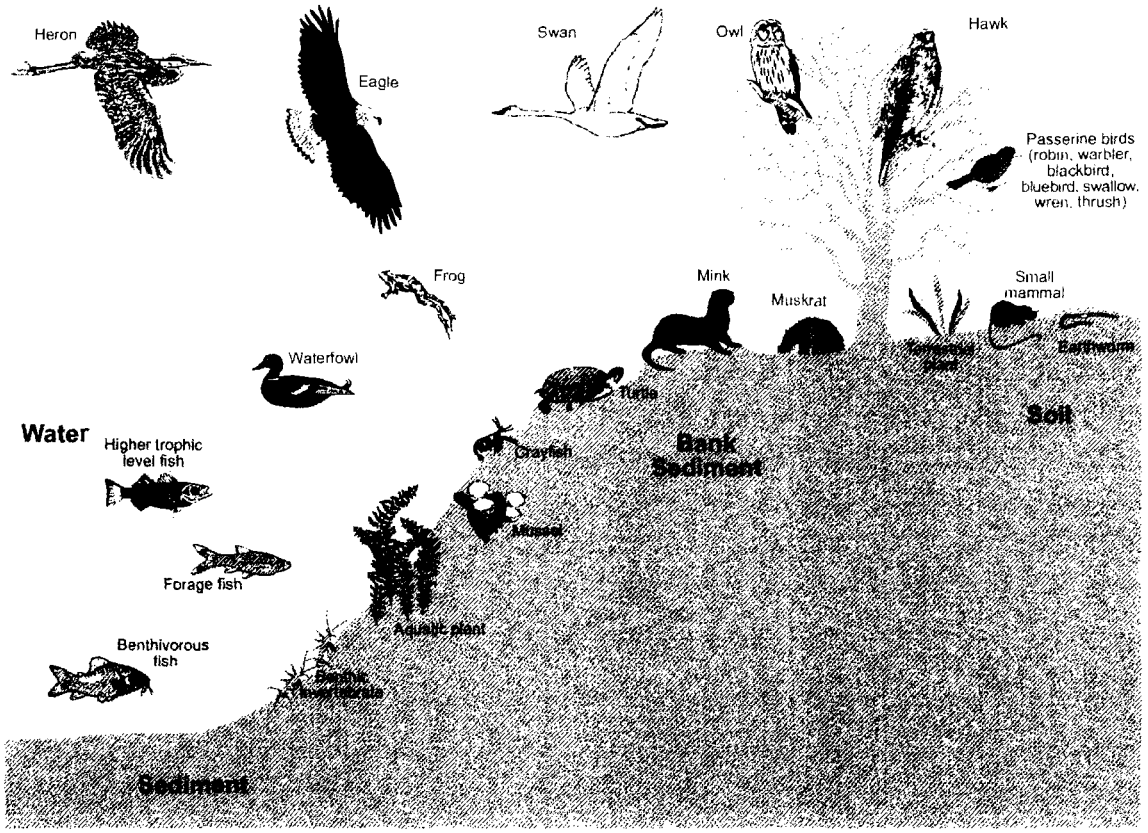

Figure 2.13. KRE natural resources in which PCB contamination has been documented (see Table 2.11 for maximum PCB concentrations measured in each resource).

Source: Swan, heron, hawk, and muskrat graphics adapted from U.S. FWS. Swan drawing by Bob Hines, heron drawing by Tom Kelley, hawk drawing by Paul Kerris, and muskrat drawing by Timothy Knepp (U.S. FWS, 2003a).

PCBs are lipophilic and thus tend to preferentially accumulate in the fatty tissue of organisms. Biota can be exposed to PCBs through routes such as dietary uptake or uptake across the gill surface. Dietary uptake is typically the primary mechanism by which animals are exposed to and subsequently bioaccumulate PCBs (Robertson and Hansen, 2001). Biota can be exposed to PCBs that are attached to the external surfaces of their food (e.g., PCBs deposited from the air onto plant tissue) and to PCBs incorporated within the food item. Ingestion of contaminated soil or sediment while feeding (either as part of the feeding strategy, such as for earthworms, or incidentally) can also contribute to the dietary intake of PCBs. Some direct uptake across the gill surface can also occur for aquatic biota (fish and invertebrates) (Niimi, 1996).

Table 2.11. Maximum total PCB concentrations measured in biota in the KRE

Biota	Maximum total PCB concentration	Source
Benthic invertebrate	1.56 mg/kg ww (composite)	Michigan State University Aquatic Toxicology Laboratory, 2003
Aquatic plant	0.047 mg/kg ww	Michigan State University Aquatic Toxicology Laboratory, 2001
Crayfish	1.93 mg/kg ww (whole body)	Michigan State University Aquatic Toxicology Laboratory, 2002a
Benthivorus fish	109.9 mg/kg ww (fillet)	MWRC, 1972a
Forage fish	38.42 mg/kg ww (fillet)	MWRC, 1972a
Higher trophic level fish	34.1 mg/kg ww (fillet)	MDNR, 1987b
Turtle	8.1 mg/kg ww (muscle)	Blasland, Bouck & Lee, 2001
Green frog	0.826 mg/kg dw (larvae)	Glennemeier and Begnoche, 2002
Waterfowl	4.8 mg/kg ww (whole body)	MDNR, 1987b
Swan	1.6 mg/kg ww (eggs)	MDNR, 1987b
Heron	44.38 mg/kg ww (eggs)	Mehne, 2000
Terrestrial plant	0.2 mg/kg ww	Michigan State University Aquatic Toxicology Laboratory, 2001
Earthworm	9 mg/kg (whole body)	MDNR, 1987d
Small mammal	3.15 mg/kg ww (whole body)	Michigan State University Aquatic Toxicology Laboratory, 2002f
Mink	12.5 mg/kg ww (liver)	Camp Dresser & McKee, 1997
Muskrat	1.18 mg/kg ww (liver)	Camp Dresser & McKee, 1997
Passerine bird	14.5 mg/kg ww (eggs)	Michigan State University Aquatic Toxicology Laboratory, 2002d
Owl	90.8 mg/kg ww (eggs)	Mehne, 2000
Hawk	27 mg/kg ww (eggs)	Mehne, 2000
Eagle	122 mg/kg ww (eggs)	Best, 2002

Table 2.11 lists the maximum PCB concentrations that have been measured in various kinds of KRE biota. The highest concentrations have been measured in bald eagle eggs (up to 122 mg/kg wet weight, or ww), owl eggs (up to 90 mg/kg ww), and in fish tissue (up to 109.9 mg/kg ww). As described in subsequent chapters of this report, these PCB concentrations in the KRE are much higher than PCB concentrations in similar biota from reference areas. The information in the table demonstrates that PCB contamination is found throughout the biota sampled in the KRE.

Figure 2.14 depicts the primary pathways through which KRE biota become exposed to PCBs in the environment. The releases of PCBs have resulted in the contamination of sediment, surface water, and soil throughout the KRE. From there, PCBs are transported through the aquatic and terrestrial food chains primarily through dietary pathways. The PCBs typically reach their highest concentrations in upper trophic level organisms, such as great-horned owl, bald eagle, and other piscivorous animals.

2.4.2 Persistence of PCBs in the KRE

PCBs are generally persistent in the environment and degrade very slowly (Erickson, 1997). Nevertheless, once released into a riverine environment such as the KRE, PCBs can be removed from the system through burial, degradation, or volatilization.

PCBs can undergo microbial biodegradation in the environment, but the extent of biodegradation, if any, is often slow and incomplete (Erickson, 1997). PCBs can undergo degradation by microbial communities in aerobic (i.e., in the presence of oxygen) and anaerobic (i.e., with oxygen absent) conditions (Abramowicz, 1990). Aerobic biodegradation of PCBs can completely break down some PCBs, producing carbon dioxide, chloride, and water (Erickson, 1997). However, aerobic biodegradation is effective only for PCBs with fewer than three chlorine atoms in their structures. In addition, there is little evidence that aerobic degradation removes any significant mass of PCBs from riverine systems such as the KRE (Erickson, 1997).

Anaerobic biodegradation of PCBs has been documented in sediments from several PCB contaminated aquatic systems (Brown and Wagner, 1990). Anaerobic microbes preferentially remove chlorine atoms from the more highly chlorinated PCBs, producing PCBs with lesser chlorination. Thus anaerobic degradation does not remove PCBs from the system, but changes the PCB congener mixture that is present. The rate of PCB anaerobic degradation is dependent on many environmental factors (Liu et al., 1996). The concentration of PCBs in sediment is one of the main factors that determines the rate of microbial degradation: degradation rates increase with increasing PCB concentration. For example, measurable dechlorination occurs in Hudson River sediments only when PCB concentrations are above approximately 30 mg/kg (U.S. EPA, 1997b). Analysis of chromatographic patterns in sediment cores from Lake Allegan and the Otsego City impoundment suggests that anaerobic degradation may be occurring in some sediments in selected areas of the Kalamazoo River (Blasland, Bouck & Lee, 2000b). However, the areas of any dechlorination in the river are most likely limited because PCB concentrations in KRE sediments are generally well below the 30 mg/kg concentration associated with measurable dechlorination in river sediments (U.S. EPA, 1997b).

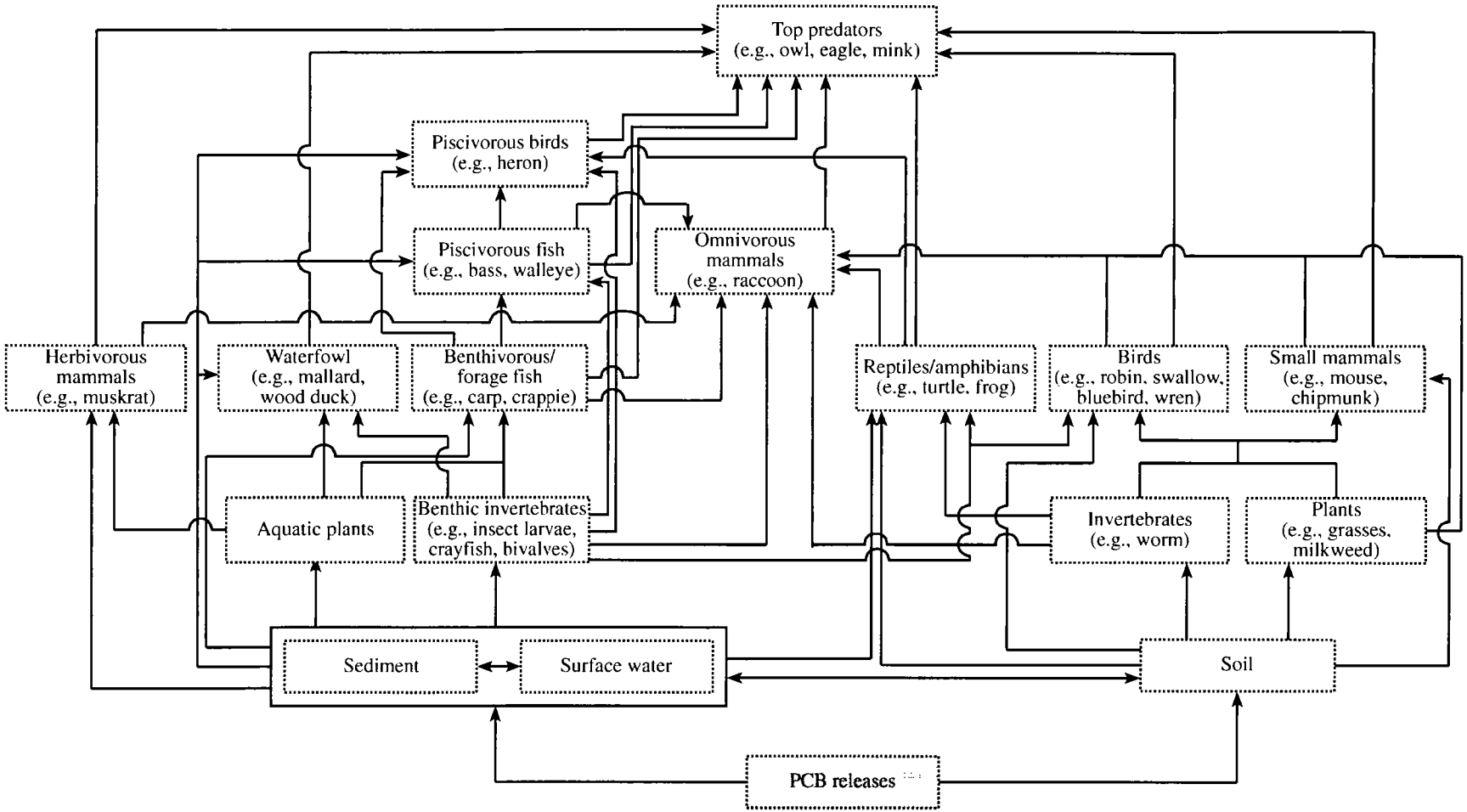

Figure 2.14. PCB exposure pathways.

PCBs can also be volatilized into the atmosphere. The lower chlorinated congeners have higher vapor pressures and tend to volatilize more readily than the higher chlorinated congeners (Mackay et al., 1992). Thus, the atmosphere tends to become enriched in the lower chlorinated congeners relative to the water column. The lower chlorinated congeners can then be carried by air currents and redistributed throughout the environment. This continuous process has produced a "global fractionation" of PCB congeners, in which the PCBs transported via air currents to northern latitudes are enriched in the lower chlorinated congeners (Muir et al., 1996). A PCB mass balance model was used to estimate the mass of PCBs lost through volatization in the open water environment of Green Bay (Wisconsin), and the model concluded that volatization accounted for a substantial portion of the PCB losses (second only to sediment burial in PCB mass removed; DePinto et al., 1994). The mass of PCBs lost through volatization in a riverine system such as the KRE is difficult to measure or predict accurately, but loss through volatization can be a significant pathway through which PCBs are removed from the KRE and subsequently transported elsewhere.

Burial in sediment is the primary mechanism by which PCBs can be removed from the water column and surface sediment environments of the KRE. Sediment burial is an important fate process for PCBs in most aquatic systems (e.g., DePinto et al., 1994; Quantitative Environmental Analysis, 1999). However, in active riverine systems such as the KRE, PCBs buried in deeper sediment layers can be re-exposed as the river bottom topography changes due to high flow events, stream channel meandering, impoundment management, or channel maintenance (Wisconsin Department of Natural Resources, 1999; Rheaume et al., 2002). Therefore, although deeper sediments contain a substantial amount of the PCBs present in the KRE, it cannot necessarily be assumed that these PCBs are in permanent isolation.

The PRPs conducted a sediment core study in 1994 in which cores were collected from Allegan City Impoundment and Kalamazoo Lake and analyzed for radionuclides and PCBs (Blasland, Bouck & Lee, 1994c). Radionuclide analysis of sediment cores can provide information on the approximate time period in which the sediments of different sections of the core were deposited. According to the dating analysis presented in Blasland, Bouck & Lee (1994c), PCB concentrations are highest in sediment deposited during the 1960s, when PCB use and direct discharge of PCBs to the river was highest. Sediment deposited after the period of peak PCB discharge to the river contains lower PCB concentrations, and concentrations have remained relatively unchanged. Blasland, Bouck & Lee (1994c) state: "The deposition rates estimated from ^{137}Cs analyses indicate that PCB deposition has been steady at least since approximately 1980," and ". . . although PCB may be present in the environment upstream, amounts being transported and deposited downstream have decreased substantially, and remained steady at relatively low concentrations." Thus the dated sediment core analysis presented in Blasland, Bouck & Lee (1994c) indicates that PCB concentrations in surficial river sediment decreased after the periods of peak releases to the river, but have since remained relatively constant. This

fact demonstrates the environmental persistence of the PCBs that have been released into the KRE.

2.5 Conclusions: PCB Releases, Pathways, and Contamination

PRP facilities released large quantities of PCBs into the KRE through the release of effluent and paper waste containing PCBs. It has been estimated that the total amount of PCBs released by Allied, Georgia-Pacific, Simpson Plainwell, and Fort James totaled between 2.2 and 4.4 million lbs (1 and 2 million kg; Rockwell Int'l Corp., 107 F. Supp. 2d 817). Additionally, PRP facilities are the predominant source of PCBs in the KRE. Recent and ongoing PCB releases include erosion and diffusion from impoundment banks, re-release from sediment and the Willow Boulevard landfill, and seeps in the Bryant Mill Pond area. These PCBs have migrated downstream in surface water, and have been deposited in instream sediments and in floodplain soils. PCBs are persistent in the environment and degrade slowly, and thus are likely to be present in the KRE for many years to come. All abiotic and biotic components of the KRE that have been measured are contaminated with PCBs released into the KRE. Active remediation that provides for the physical removal of the PCBs from the KRE would ensure that the PCB exposure pathways are permanently eliminated.

3. Injuries to Surface Water

Surface water resources are defined in the DOI regulations to include both surface water and sediments suspended in water or lying on the bank, bed, or shoreline [43 C.F.R. § 11.14(pp)]. This chapter presents the Stage I Injury Assessment for surface water, and Chapter 4 presents the assessment for bed sediment. Bed sediment is addressed in a separate chapter for several reasons: (1) there is a large amount of data specific to sediments, (2) sediments can be a principal and ongoing exposure pathway to other natural resources, and (3) some PCB cleanup actions for the Kalamazoo River may focus on sediments.

Ecosystem services provided by surface water include habitat for fish, migratory birds, benthic macroinvertebrates, and aquatic, semiaquatic, and amphibious animals; water, nutrient and sediment transport to riparian vegetation; nutrient cycling; geochemical exchange processes; primary and secondary productivity and transport of energy (food) to downstream and downgradient organisms; growth media for aquatic and wetland plants; and a migration corridor. Human use services may include drinking water, swimming, boating, industrial water supply, other water-based recreation, and assimilative capacity (i.e., the ability of a resource to "absorb low levels of [contaminants] without exceeding standards or without other effects)" [51 Fed. Reg. 27716, Aug. 1, 1986].

3.1 Injury Definitions

In this chapter, injuries to surface water resources are determined using the following injury definitions:

- Concentrations and duration of substances in excess of drinking water standards as established by Sections 1411-1416 of the Safe Drinking Water Act (SDWA), or by other federal or state laws or regulations that establish such standards for drinking water, in surface water that was potable before the hazardous substance release [43 C.F.R. § 11.62(b)(1)(i)].

- Concentrations and duration of substances in excess of applicable water quality criteria established by Section 304(a)(1) of the CWA, or by other federal or state laws or regulations that establish such criteria, in surface water that before the . . . release met the criteria and is a committed use . . . as a habitat for aquatic life, water supply, or recreation. The most stringent criterion shall apply when surface water is used for more than one of these purposes [43 C.F.R. § 11.62(b)(1)(iii)].

In addition, injury to surface water can be evaluated with the following definition:

- Concentrations and duration of substances sufficient to have caused injury . . . to ground water, air, geologic, or biological resources, when exposed to surface water; suspended sediments; or bed, bank, or shoreline sediments [43 C.F.R. § 11.62(b)(1)(v)].

However, application of this injury definition requires evaluation of other natural resources, which is not included in this chapter. Injury to bed sediment is assessed in Chapter 4, and injuries to aquatic biota are assessed in Chapters 5 and 6.

3.2 Stage I Injury Assessment Approach

Table 3.1 outlines the approach taken in this chapter to assess injury to surface water, which consists of comparing measured concentrations of PCBs in surface water to appropriate water quality standards and criteria. As described in Chapter 1, the focus is on available surface water data from approximately the past 20 years.

Table 3.1. Approaches to evaluate injury to surface water

Injury definition	Stage I injury assessment approach	Chapter section
Concentrations and duration of substances in excess of drinking water standards . . . in surface water that was potable before the release [43 C.F.R. § 11.62(b)(1)(i)]	Compare measured surface water PCB concentrations to state and federal drinking water standards.	3.3
Concentrations and duration of substances in excess of applicable water quality criteria or standards, in surface water that before the . . . release met the criteria and is a committed use . . . as a habitat for aquatic life, water supply, or recreation [43 C.F.R. § 11.62(b)(1)(iii)]	Compare measured surface water PCB concentrations to federal water quality criteria and state water quality standards. Evaluate committed uses.	3.4

3.3 Drinking Water Standard Exceedences

3.3.1 Data sources

The following sources of data on PCB concentrations in surface water are used in this evaluation:

- Surface water PCB concentrations measured by MDNR in Portage Creek and the Kalamazoo River (including upstream reference areas) in 1985 to 1987 (MDNR, 1987b)

- Surface water PCB concentrations measured by Blasland, Bouck & Lee in Portage Creek and the Kalamazoo River (including upstream reference areas) in 2000 and 2001 (Blasland, Bouck & Lee, 2001; methods in Blasland, Bouck & Lee, 2000c).

Surface water samples discussed in this report were analyzed as unfiltered samples for PCBs. For this evaluation, samples in which no PCBs were detected were plotted as one half of the detection limit and identified with a hollow symbol.

The PCB surface water sampling locations in Portage Creek and the Kalamazoo River were organized by the Trustees into reaches (Table 3.2 and Figure 3.1). These reaches are based on the reaches used in the draft Remedial Investigation/Feasibility Study (RI/FS) prepared by the PRPs (Blasland, Bouck & Lee, 2000a).

Table 3.2. Reach designations for surface water samples

Reach designation	Reach description	Reference or assessment
Kalamazoo River (upstream)	Upstream of Morrow Dam	Reference
Portage Creek (upstream)	Portage Creek upstream of PRPs	Reference
Reach A1	Morrow Dam to Portage Creek confluence	Assessment
Portage Creek (downstream)	Portage Creek downstream of PRPs	Assessment
Reach A2	Portage Creek confluence to Main Street, Plainwell	Assessment
Reach B	Main Street, Plainwell to Plainwell Dam	Assessment
Reach C[a]	Plainwell Dam to Otsego City Dam	Assessment
Reach D	Otsego City Dam to Otsego Dam	Assessment
Reach E[a]	Otsego Dam to Trowbridge Dam	Assessment
Reach F	Trowbridge Dam to the Allegan City line	Assessment
Reach G	Allegan City line to Allegan City Dam	Assessment
Reach H	Allegan City Dam to Lake Allegan Dam	Assessment
Reach I	Lake Allegan Dam to Lake Michigan	Assessment

a. No water samples were available for RI/FS reaches C and E.

3.3.2 Regulatory criteria and standards

Table 3.3 lists applicable PCB drinking water standards that have been established to protect drinking water supplies and that may be used to evaluate injury to surface waters, as defined in 43 C.F.R. § 11.62(b)(1)(i). The SDWA establishes a Maximum Contaminant Level (MCL) for PCBs of 0.5 µg/L [40 C.F.R. § 141]. This value is the maximum permissible concentration of

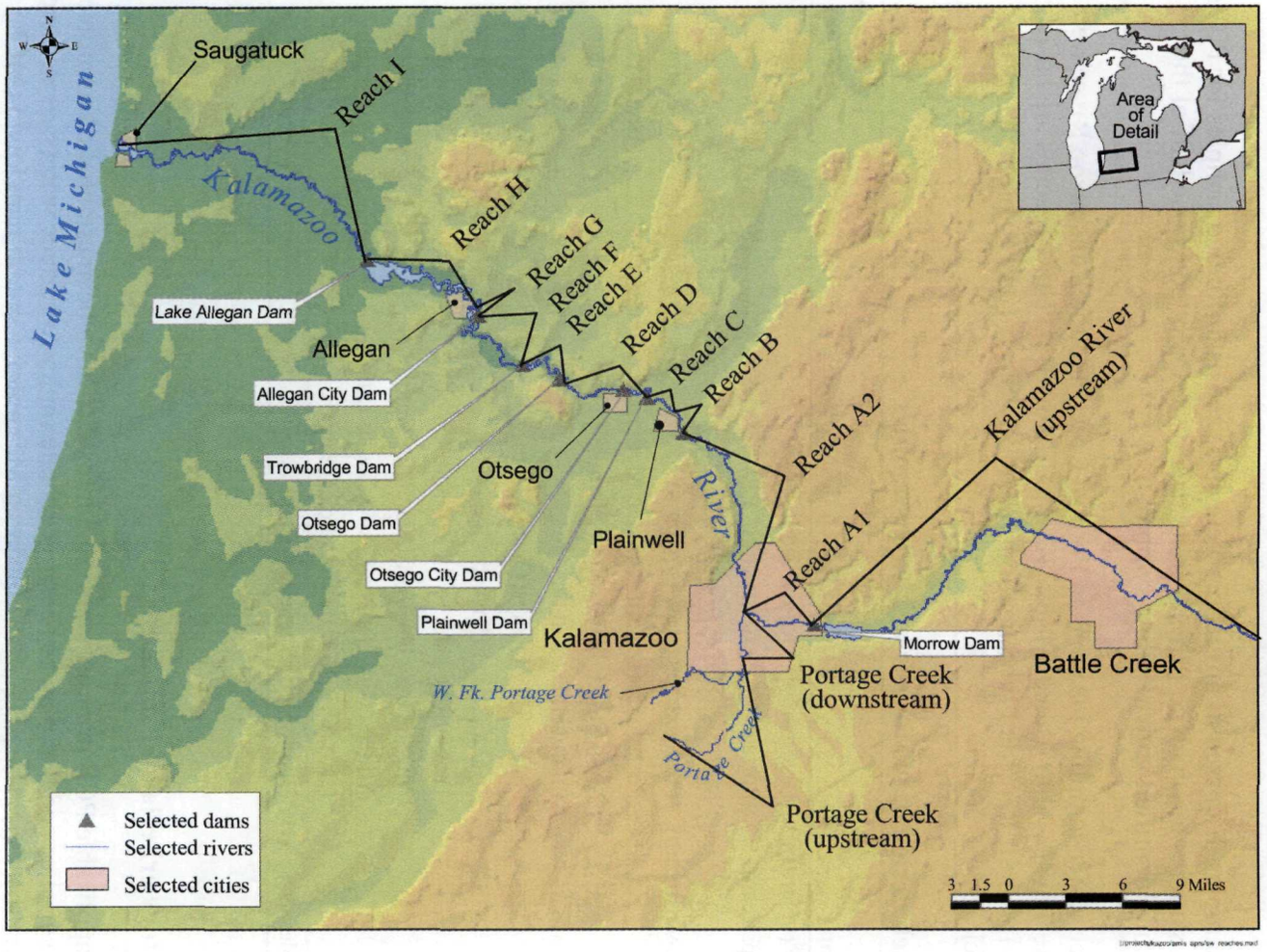

Figure 3.1. Locations of reach designations for surface water samples.

Table 3.3. Drinking water standards for determining injury to surface water

Source	Drinking water standard (µg/L)
SDWA MCL[a]	0.5
Michigan Safe Drinking Water Act MCL[b]	0.5

a. 40 C.F.R. § 141.
b. Michigan Act 399 of 1976, Section 325.1006.

PCBs in water which is delivered to any user of a public water system. Additionally, EPA established a Maximum Contaminant Level Goal (MCLG) for PCBs of 0 (U.S. EPA, 1995). This value is a nonenforceable concentration that is protective of human health effects and allows an adequate margin of safety. The State of Michigan has also incorporated MCLs for contaminants by reference to the federal SDWA, and thus the state MCL for PCBs is 0.5 µg/L (Michigan Act 399 of 1976, Section 325.1006). The state and federal MCL of 0.5 µg/L PCBs will be used to determine injury to surface water according to this definition of injury.

3.3.3 Results

Exceedences of drinking water standards

No surface water PCB concentrations measured in Portage Creek or the Kalamazoo River downstream of PRP facilities in 1985 to 1987 (Figure 3.2) or 2000 to 2001 (Figure 3.3) exceeded the state and federal MCL of 0.5 µg/L. Therefore, the Trustees conclude that surface water is not injured according to the injury definition described in 43 C.F.R. § 11.62(b)(1)(i).

Nevertheless, PCB concentrations were much more elevated downstream of PRP facilities than in upstream reference locations in 1985 to 1987 (Figure 3.2) and 2000 to 2001 (Figure 3.3). For example, the maximum PCB concentration measured downstream of PRP facilities in 1985-1987, 0.34 µg/L, was 20 times higher than the maximum concentration observed upstream of PRP facilities. The maximum PCB concentration measured downstream of PRP facilities in 2000-2001, 0.22 µg/L, was 29 times higher than the highest concentration observed upstream of PRP facilities (Figure 3.3).

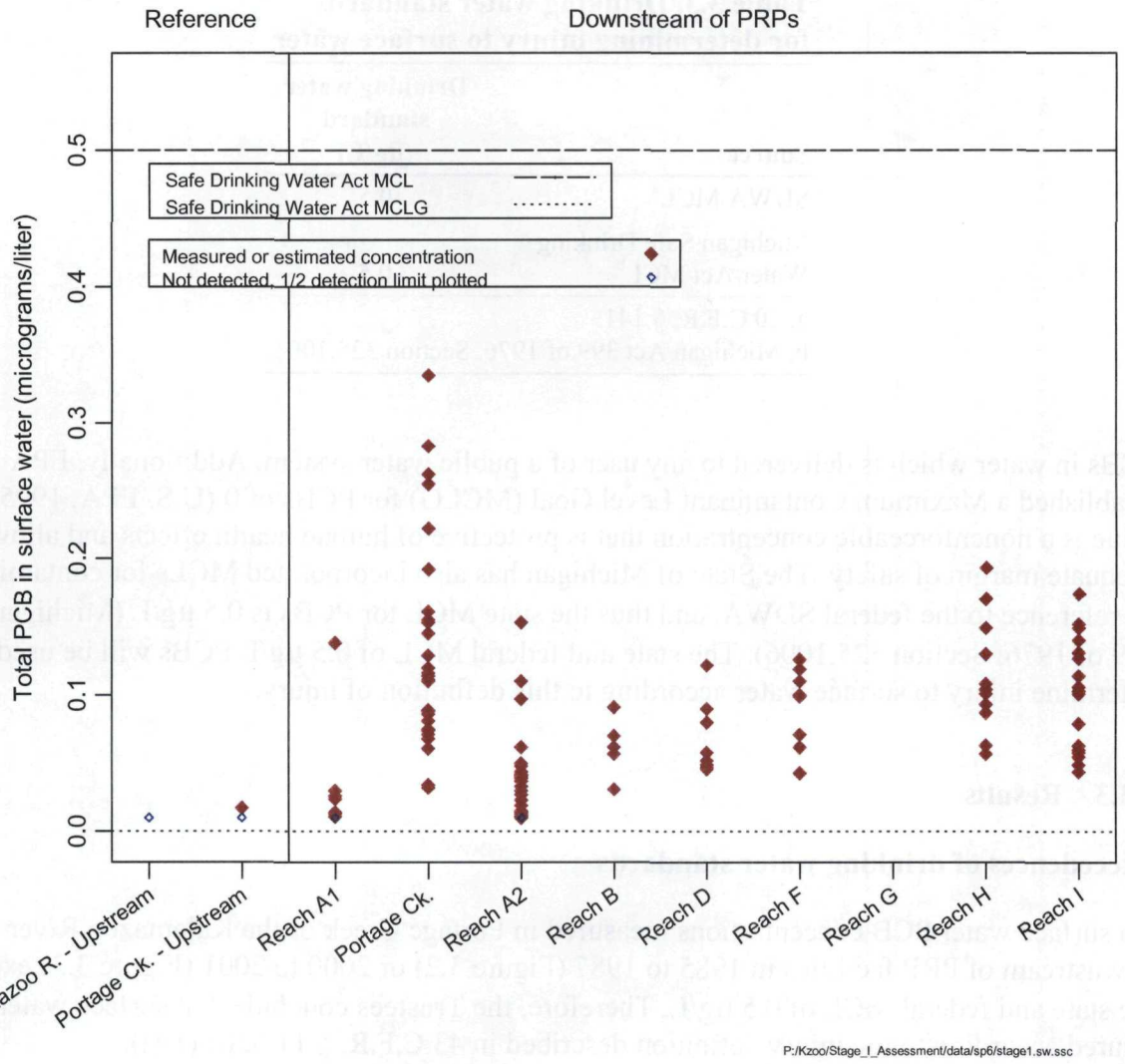

Figure 3.2. Surface water total PCB concentrations in Portage Creek and the Kalamazoo River from 1985 to 1987 compared to drinking water standards.

Source: MDNR, 1987b.

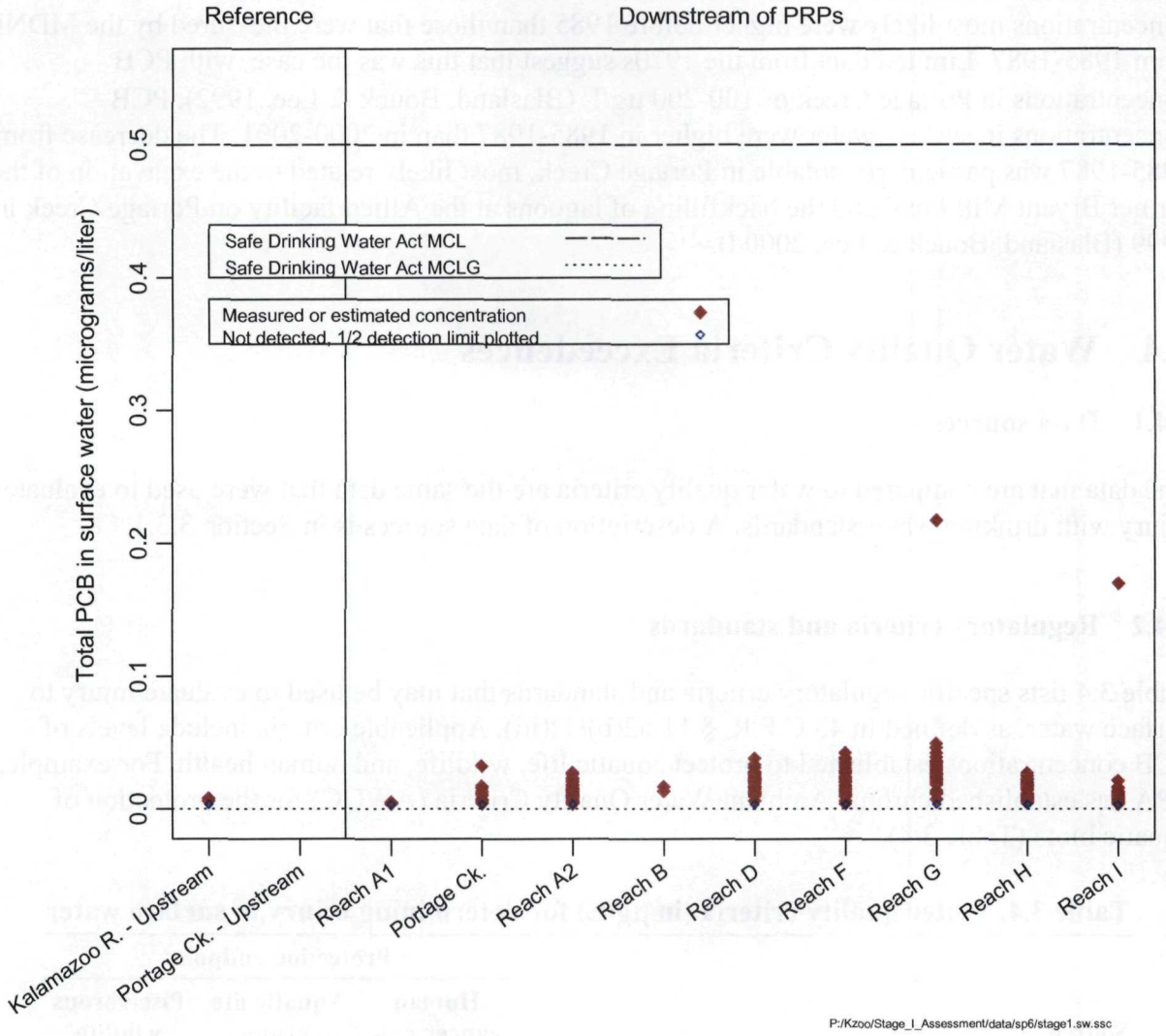

Figure 3.3. Surface water total PCB concentrations in Portage Creek and the Kalamazoo River from 2000 and 2001 compared to drinking water standards.

Source: Blasland, Bouck & Lee, 2001.

Because of the timing of PCB uses and releases from PRP facilities, surface water PCB concentrations most likely were higher before 1985 than those that were measured by the MDNR from 1985-1987. Limited data from the 1970s suggest that this was the case, with PCB concentrations in Portage Creek of 100-200 µg/L (Blasland, Bouck & Lee, 1992). PCB concentrations in surface water were higher in 1985-1987 than in 2000-2001. The decrease from 1985-1987 was particularly notable in Portage Creek, most likely related to the excavation of the former Bryant Mill Pond and the backfilling of lagoons at the Allied facility on Portage Creek in 1999 (Blasland, Bouck & Lee, 2000d).

3.4 Water Quality Criteria Exceedences

3.4.1 Data sources

The data that are compared to water quality criteria are the same data that were used to evaluate injury with drinking water standards. A description of data sources is in Section 3.3.1.

3.4.2 Regulatory criteria and standards

Table 3.4 lists specific regulatory criteria and standards that may be used to evaluate injury to surface water, as defined in 43 C.F.R. § 11.62(b)(1)(iii). Applicable criteria include levels of PCB concentrations established to protect aquatic life, wildlife, and human health. For example, EPA has established chronic Ambient Water Quality Criteria (AWQC) for the protection of aquatic biota (Table 3.4).

Table 3.4. Water quality criteria (in µg/L) for determining injury to surface water

	Protection endpoint		
Source	**Human cancer risk**	**Aquatic life (chronic)**	**Piscivorous wildlife**
EPA AWQC[a]		0.014	
National Toxics Rule[b]	0.00017	0.014	
Michigan Water Quality Standard (Rule 323.1057)[c]	0.000026		0.00012
Great Lakes Water Quality Guidance (GLWQG) (40 C.F.R.Part 132)[d]	0.000026		0.00012

a. U.S. EPA, 1999.
b. 57 Fed. Reg. 60915; 63 Fed. Reg. 61181-61196; 62 Fed. Reg. 42159-42208.
c. MDEQ, 1994b.
d. 62 Fed. Reg. 11723-11731; 62 Fed. Reg. 52921-52924.

DOI regulations indicate that "the most stringent criterion shall apply when surface water is used for more than one of these purposes" (habitat for aquatic life, water supply, or recreation) [43 C.F.R. § 11.62(b)(1)(iii)]. For this Stage I Assessment, PCB concentrations in the Kalamazoo River and Portage Creek are compared to the Michigan Water Quality Standards for human cancer risk of 0.000026 µg/L and for protection of piscivorous wildlife of 0.00012 µg/L, which are equivalent to the GLWQG values. Additionally, surface water data are compared to the EPA chronic AWQC for aquatic life of 0.014 µg/L, which is equivalent to the National Toxics rule criteria.

3.4.3 Results

Exceedences of PCB standards

PCB concentrations in samples collected in Portage Creek and the Kalamazoo River downstream of PRP facilities exceed the Michigan Water Quality Standard for the protection of wildlife (Figures 3.4 and 3.5) and the Michigan Water Quality Standard for human cancer risk (not plotted). Additionally, the EPA chronic AWQC was exceeded downstream of PRP facilities (Figures 3.4 and 3.5).

PCB concentrations measured in 1985-1987 were highest in Portage Creek downstream of PRP facilities (Figure 3.4). In this reach, the maximum observed concentration of 0.34 µg/L is 2,800 times higher than the Michigan Water Quality Standard for the protection of wildlife, 13,000 times higher than the Michigan Water Quality Standard for human cancer risk, and 24 times higher than the EPA chronic AWQC. Concentrations in mainstem reaches of the Kalamazoo River were also as much as four orders of magnitude greater than the Michigan Water Quality Standards. Concentrations did not decrease in downstream reaches and were up to 0.17 µg/L between Lake Allegan Dam and Lake Michigan (Reach I). In contrast, PCBs were detected in 1 of 26 samples (4%) collected in upstream reference reaches of the Kalamazoo River and Portage Creek at a concentration of 0.02 µg/L.

Although concentrations in Portage Creek and the Kalamazoo River downstream of PRP facilities measured in 2000 and 2001 were lower than those measured in 1985-1987, concentrations remained several times greater than water quality criteria (Figure 3.5). The Michigan Water Quality Criteria for the protection of wildlife and human health were exceeded in all assessment reaches in Portage Creek and the Kalamazoo River, and the EPA AWQC was exceeded in all of the assessment reaches, except for Reach A1. In reaches D, F, and G, the percentages of all samples exceeding the EPA AWQC were 58%, 66%, and 73%, respectively. The maximum measured concentration, in Reach G, was 0.22 µg/L, which is 1,800 times higher than the Michigan Water Quality Standard for the protection of wildlife, 8,300 times higher than the Michigan Water Quality Standard for human cancer risk, and 16 times higher than the EPA

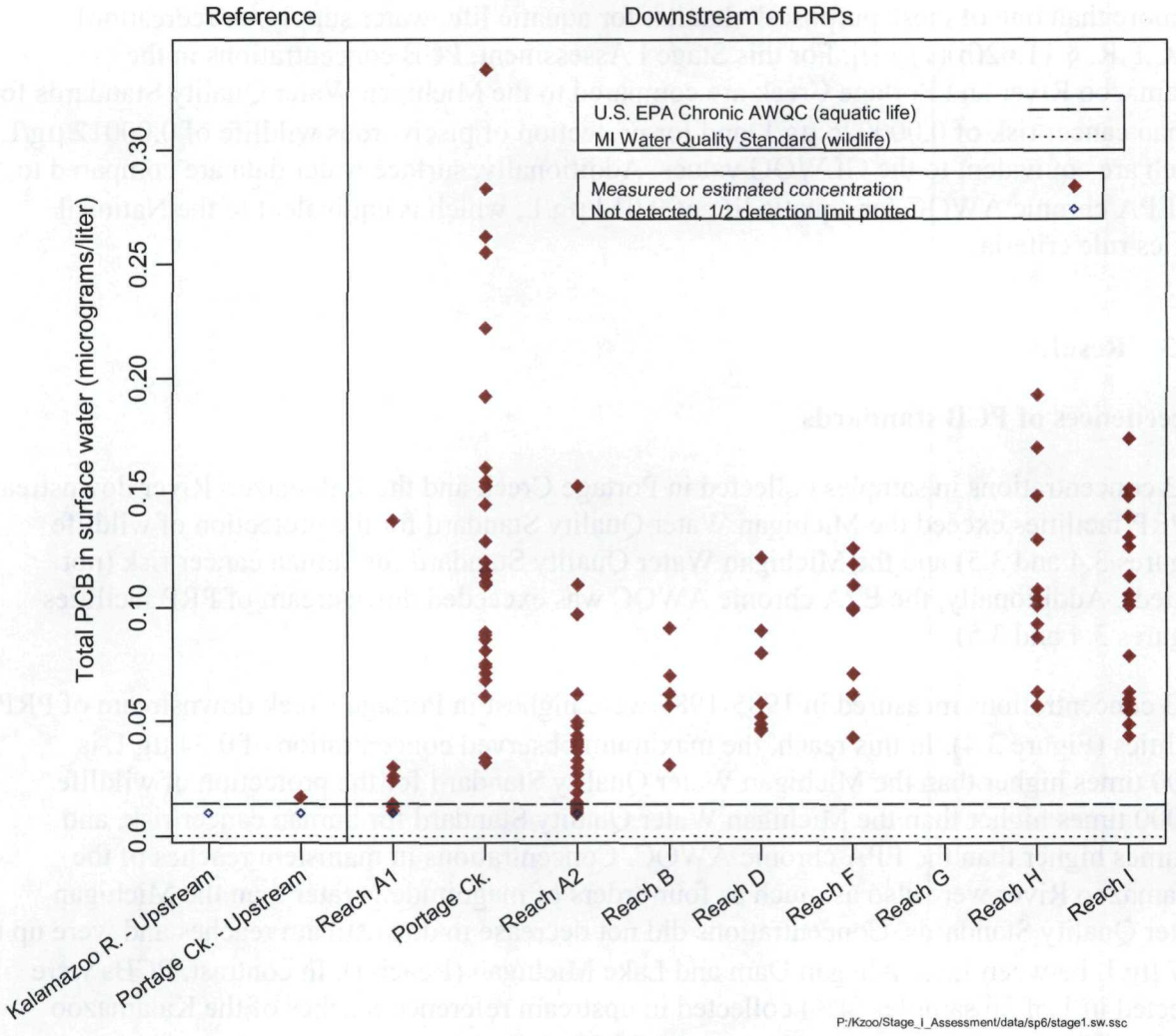

Figure 3.4. Surface water total PCB concentrations in Portage Creek and the Kalamazoo River from 1985 to 1987 compared to water quality criteria.

Source: MDNR, 1987b.

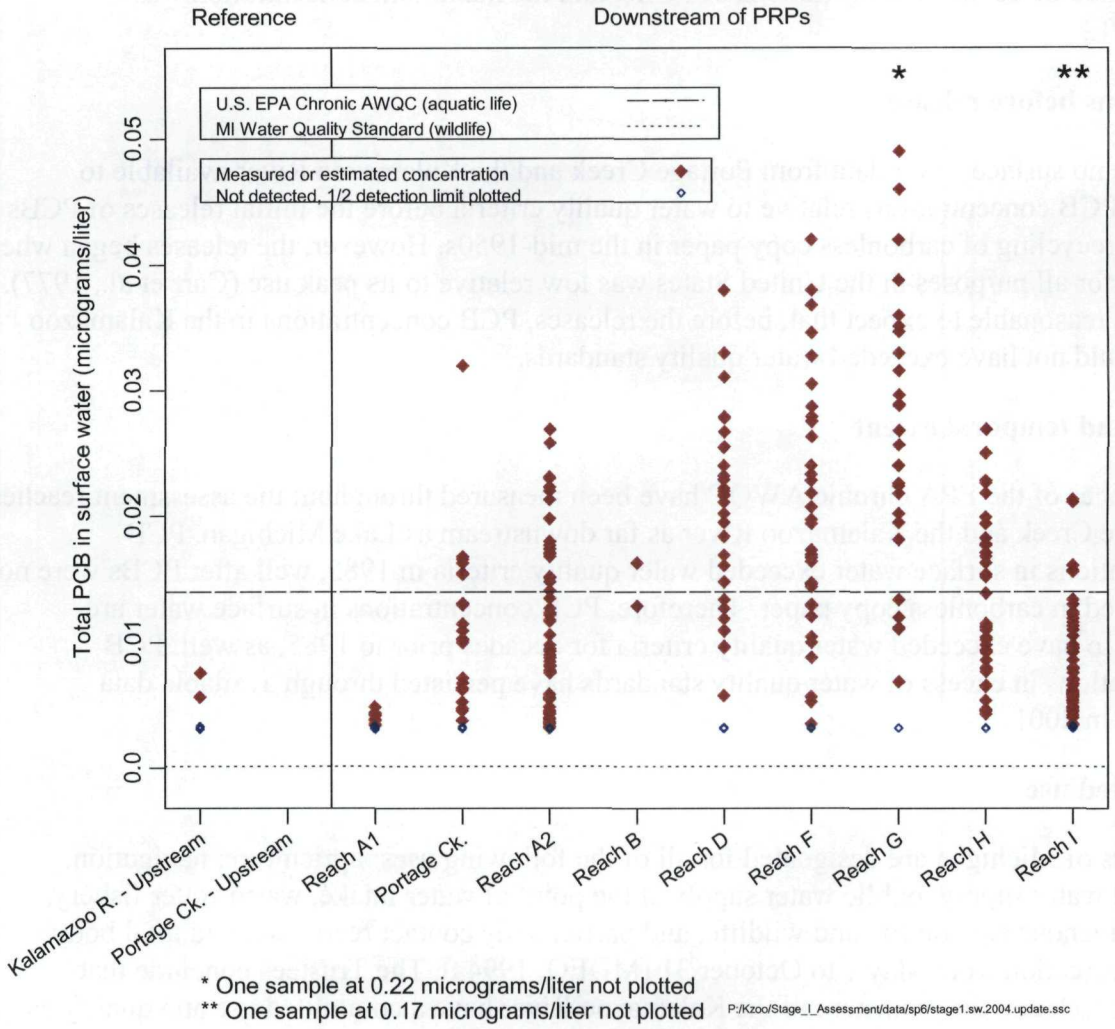

Figure 3.5. Surface water total PCB concentrations in Portage Creek and the Kalamazoo River in 2000 and 2001 compared to water quality criteria.

Source: Blasland, Bouck & Lee, 2001.

chronic AWQC. Only 2 of 33 samples (6%) from the Kalamazoo River upstream of PRP facilities had detectable concentrations of PCBs, and the maximum concentration was 0.008 µg/L.

Conditions before release

There are no surface water data from Portage Creek and the Kalamazoo River available to evaluate PCB concentrations relative to water quality criteria before the initial releases of PCBs from the recycling of carbonless copy paper in the mid-1950s. However, the releases began when PCB use for all purposes in the United States was low relative to its peak use (Carr et al., 1977). Thus it is reasonable to expect that, before the releases, PCB concentrations in the Kalamazoo River would not have exceeded water quality standards.

Spatial and temporal extent

Exceedences of the EPA chronic AWQC have been measured throughout the assessment reaches of Portage Creek and the Kalamazoo River as far downstream as Lake Michigan. PCB concentrations in surface water exceeded water quality criteria in 1985, well after PCBs were no longer used in carbonless copy paper. Therefore, PCB concentrations in surface water are expected to have exceeded water quality criteria for decades prior to 1985, as well. PCB concentrations in excess of water quality standards have persisted through available data collected in 2001.

Committed use

All waters of Michigan are designated for all of the following uses: agriculture, navigation, industrial water supply, public water supply at the point of water intake, warm water fishery, other indigenous aquatic life and wildlife, and partial body contact recreation and total body contact recreation from May 1 to October 31 (MDEQ, 1994a). The Trustees conclude that Portage Creek and the mainstem of the Kalamazoo River have a committed use and qualify as injured under 43 C.F.R. § 11.62(b)(1)(iii).

3.5 Conclusions

Available data from the mid-1980s and from 2000 to 2001 indicate that surface water PCB concentrations in Portage Creek and the Kalamazoo River downstream of PRP facilities are much higher than those observed in upstream reference locations (see Figures 3.2-3.5). PCBs have most likely been elevated since the initial releases from the PRP facilities, and concentrations were probably much higher in the past than those measured in 1985.

Surface water PCB concentrations in Portage Creek and the Kalamazoo River do not exceed the Safe Drinking Water Act MCL of 0.5 µg/L. The Trustees thus conclude that surface water is not injured according to the injury definition in 43 C.F.R. § 11.62(b)(1)(i).

However, surface water PCB concentrations downstream of PRP facilities have exceeded applicable water quality criteria established by the State of Michigan and EPA for human cancer risk and for the protection of aquatic life and piscivorous wildlife by nearly an order of magnitude. Based on the sources and timing of the releases of PCBs from PRP facilities, it is highly unlikely that PCBs would have been present before the initial releases of PCBs from the recycling of carbonless copy paper in the mid-1950s from the PRP facilities, and thus it is reasonable to conclude that water did not exceed criteria before this time. Additionally, the Kalamazoo River and Portage Creek have designated committed uses which are relevant to the exceedence of water quality criteria. The Trustees thus conclude that surface water is and has been injured according to the injury definition in 43 C.F.R. § 11.62(b)(1)(iii).

4. Injuries to Sediment

As noted in Chapter 3, surface water resources are defined in the DOI regulations to include both surface water and sediments suspended in water or lying on the bank, bed, or shoreline [43 C.F.R. § 11.14(pp)]. Injury to surface water was discussed in Chapter 3, and injury to bed sediments is discussed in this chapter.

Ecosystem services provided by sediment include habitat for fish, benthic macroinvertebrates, semiaquatic and amphibious animals, and aquatic and riparian vegetation; hydrologic flux; and nutrient and energy cycling.

4.1 Injury Definitions

In this chapter, injuries to sediment resources are determined using the following injury definition:

- Concentrations . . . of substances sufficient to have caused injury . . . to groundwater, air, geologic, or biological resources when exposed to . . . sediments [43 C.F.R. § 11.62(b)(1)(v)].

4.2 Stage I Injury Assessment Approach

Table 4.1 outlines the approaches taken in this chapter to assess injury to sediment. Concentrations of PCBs measured in instream sediment are compared to appropriate toxicity reference values associated with adverse effects in benthic invertebrates and mink.

4.3 Comparison to Concentrations Affecting Benthic Invertebrates

Benthic invertebrates are invertebrates that live or feed on the bottom of aquatic habitats. Examples include clams, snails, mussels, and the larval forms of some insects (e.g., dragonflies, midges, mayflies). They are vitally important in the aquatic food chain, playing essential roles in energy and nutrient transfer from primary producers such as algae and phytoplankton to predatory fish and as decomposers. Benthic invertebrates can be exposed to PCBs in sediment via ingestion or accumulation across the gills. In this section, the potential for PCBs in the sediment of Portage Creek and the Kalamazoo River to injure benthic invertebrates is assessed.

Table 4.1. Approaches to evaluate injury to sediment

Injury definition	Stage I injury assessment approach	Chapter section
Concentrations of . . . substances sufficient to have caused injury . . . to ground water, air, geologic, biological or surface water resources that are exposed to . . . sediments [43 C.F.R. § 11.62(b)(1)(v)]	Compare surface sediment concentrations to consensus-based sediment effect concentrations for benthic invertebrates.	4.3
	Compare surface sediment concentrations to site specific thresholds for causing risk to *mink via food chain exposure.*	4.4

4.3.1 Data sources

Data used to evaluate injury to benthic invertebrate resources from exposure to sediment are drawn from the following source:

- Sediment PCB concentrations in samples collected from 1993 to 2000 by Blasland, Bouck & Lee (2001).

Sediment samples were collected in reference and assessment sites in Portage Creek and the Kalamazoo River in 1993, 1994, 1997, 1999, and 2000 and analyzed for PCBs as Aroclors (Blasland, Bouck & Lee, 2001). The methods used are outlined in the RI/FS Draft Technical Memorandum 12 (Blasland, Bouck & Lee, 1994c), the RI/FS Draft Memorandum 10 (Blasland, Bouck & Lee, 1994a), and Appendix S-1 of the Supplement to the Kalamazoo River RI/FS (Blasland, Bouck & Lee, 2000c). All concentrations in sediment are expressed on a dry weight basis, unless specified otherwise.

The sediment data presented in this evaluation include only the surface sections of sediment cores and surface grab samples, because surface sediment concentrations are the most biologically relevant to benthic organisms. A depth-weighted average concentration over all sampled segments from 0 to 6 inches of each core was calculated by multiplying the PCB concentration in each sample of a core from 0 to 6 inches by the core length of that sample, summing all such products for the core, and dividing this sum by total core length of these samples (typically 6 inches). The majority of cores (95%) had sampled segments representative of the entire 0-6 in. range. The remaining cores either did not extend as deep as 6 inches (such as surface grab samples) or did not have a complete surface section. In these cases, a depth-weighted average concentration was calculated using the available portions of these cores between 0 and 6 inches. For this evaluation, sampled segments in which no PCBs were detected were assigned a value of one half of the reported detection limit.

Only data for which coordinate information was available are included in this evaluation. The PCB sediment sampling locations in Portage Creek and the Kalamazoo River were organized by the Trustees into reaches (Table 4.2 and Figure 4.1). These reaches are based on the reaches used in the draft RI/FS prepared by the PRPs (Blasland, Bouck & Lee, 2000a). The approximate lengths of these reaches ranges from 1.6 to 25.2 miles.

Table 4.2. Reach designations for sediment samples

Reach designation	Approximate length (miles)	Reach description	Reference or assessment
Kalamazoo River (upstream)	NA	Upstream of Morrow Dam	Reference
Reach A1 (upstream)[a]	2.7	Morrow Dam to Georgia Pacific A-Site	Reference
Reach A1 (downstream)[a]	2.0	Georgia Pacific A-Site to Portage Creek confluence	Assessment
Portage Creek	3.1	Portage Creek (downstream of Allied)	Assessment
Reach A2	14.2	Portage Creek confluence to Main Street, Plainwell	Assessment
Reach B	1.8	Main Street, Plainwell to Plainwell Dam	Assessment
Reach C	1.6	Plainwell Dam to Otsego City Dam	Assessment
Reach D	3.3	Otsego City Dam to Otsego Dam	Assessment
Reach E	4.5	Otsego Dam to Trowbridge Dam	Assessment
Reach F	7.1	Trowbridge Dam to the Allegan City line	Assessment
Reach G	1.7	Allegan City line to Allegan City Dam	Assessment
Reach H	10.0	Allegan City Dam to Lake Allegan Dam	Assessment
Reach I	25.2	Lake Allegan Dam to Lake Michigan	Assessment

a. The spatial distribution of the available sediment data allowed Reach A1 to be separated into A1 upstream of all PRP facilities and A1 downstream of any PRP facilities.

4.3.2 Toxicity reference value derivation

To evaluate the potential for PCBs in sediments to cause toxicity to benthic macroinvertebrates, several different regulatory agencies or research groups have developed sediment effect concentrations (SECs). These SECs provide a means of evaluating the potential for contaminated sediment to cause toxicity to sediment-dwelling aquatic biota. However, no national or state regulatory criteria or standards have been developed for sediments.

MacDonald et al. (2000) reviewed and assembled published SECs for PCBs that were empirically based, relying on databases of sediment contamination and effects to invertebrates. The SECs assembled by MacDonald et al. (2000) differ in the underlying databases used to develop them, the statistical approaches employed to derive SECs from the databases, and the interpretations of the results of the statistical approaches.

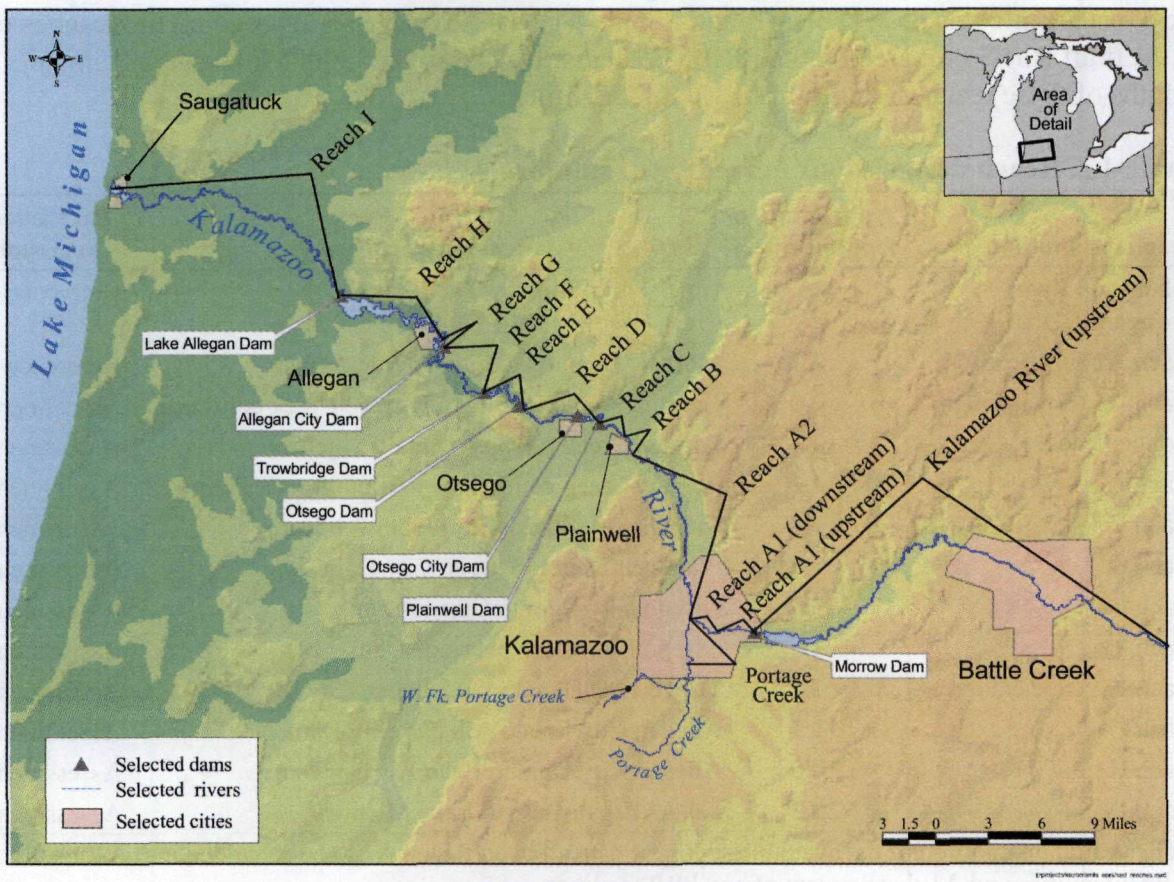

Figure 4.1. Locations of reach designations for sediment samples.

MacDonald et al. (2000) then classified the SECs into three categories: a threshold effect concentration (TEC), a midrange effect concentration (MEC), and an extreme effect concentration (EEC). TECs are intended to identify contaminant concentrations below which harmful effects to sediment-dwelling organisms are not expected to occur. The TECs include selected published minimal effect thresholds, lowest effect levels, and screening level concentrations, and they are intended to protect 85 to 90% of sediment-dwelling organisms. The MECs are intended to identify contaminant concentrations above which harmful effects on sediment-dwelling organisms are expected to occur frequently. The MECs include selected published effect range median concentrations, probable effect levels, moderate apparent effect thresholds, and probable apparent effects thresholds. The EECs are intended to identify contaminant concentrations above which harmful effects are usually or always observed. The

EECs include selected published highest apparent effect thresholds, toxic effect thresholds, and severe effect levels.

Using the geometric means of the three categories of SECs, MacDonald et al. (2000) derived "consensus-based" SECs and evaluated their predictive ability. Of the samples with PCB concentrations less than the TEC, 84% were not toxic to invertebrates. Of the samples with PCB concentrations greater than the MEC and EEC, 68% and 83%, respectively, were toxic to invertebrates. Thus, MacDonald et al. (2000) concluded that the consensus-based SECs are effective at predicting the toxicity of PCBs in freshwater sediments.

The consensus MEC and EEC are used in this Stage I Assessment as potential injury thresholds. Table 4.3 presents the PCB MEC and EEC values, and includes the TEC for comparison.

Table 4.3. SECs for invertebrate exposure to PCBs in freshwater sediment

Name	Definition	Basis	Sediment PCB concentration (mg/kg dry wt)
Consensus TEC	Concentration below which adverse effects are not likely to occur	Geometric mean of 12 TECs	0.04
Consensus MEC	Concentration above which adverse effects are frequently observed	Geometric mean of 12 MECs	0.4
Consensus EEC	Concentration above which adverse effects are usually or always observed	Geometric mean of 5 EECs	1.7

Source: MacDonald et al., 2000.

4.3.3 Results

Depth-weighted mean surface sediment concentrations are higher downstream of PRP facilities than in upstream reference reaches (Figure 4.2). The maximum measured depth-weighted mean surface sediment concentration in the assessment reaches, 117 mg/kg in Reach B, is 63 times higher than any concentration measured in samples taken upstream of PRP facilities.

The consensus sediment effect concentrations are exceeded more often and by a greater magnitude downstream of PRP facilities than upstream, and are exceeded in all of the assessment reaches. Concentrations in 37% of all surface sediment samples collected from assessment reaches exceed the MEC and 17% exceed the EEC. In upstream reference reaches, 18% of samples are higher than the MEC and 3% are higher than the EEC. The percentage of surface samples in assessment reaches which exceed the MEC ranges from 18.1% in Reach A1 to 85.7%

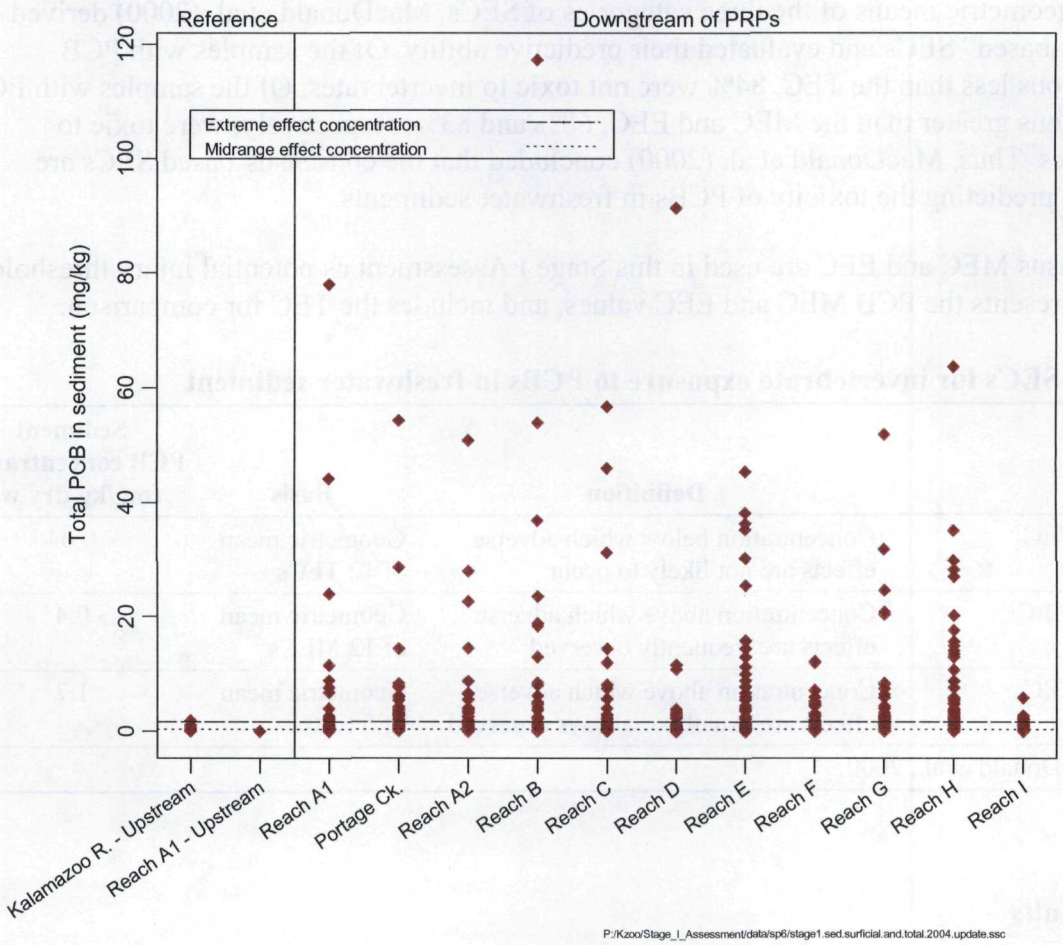

Figure 4.2. Depth-weighted mean PCB concentrations in surficial sediment from cores collected in Portage Creek and the Kalamazoo River compared to consensus-based sediment effect concentrations.

Sources: MacDonald et al., 2000; Blasland, Bouck & Lee, 2001.

in Portage Creek and the percentage which exceed the EEC ranges from 4.4% in Reach I to 53% in Reach H (Table 4.4). In most assessment reaches, some samples have PCB concentrations greater than ten times the EEC. Therefore, because of the frequency and magnitude of exceedences of the PCB MEC and EEC, PCB concentrations in sediment most likely are sufficient to cause injury to benthic invertebrates.

Table 4.4. Exceedences of consensus-based MEC and EEC by depth-weighted mean PCB concentrations in surficial sediment of Portage Creek and the Kalamazoo River

Reach	Number of samples	PCB concentration (mg/kg)[a]	% exceeding MEC[b]	% exceeding MEC by > 10 times	% exceeding EEC[c]	% exceeding EEC by > 10 times
Kalamazoo River – upstream	22	0.39 (0.03-1.88)	31.8	0	4.55	0
Reach A1 – upstream	17	0.03 (0.02-0.05)	0	0	0	0
Reach A1	166	1.33 (0.02-78)	18.1	4.22	8.43	1.81
Portage Creek	49	4.03 (0.07-54.3)	85.7	20.4	44.9	4.08
Reach A2	282	0.79 (0.02-50.8)	19.5	3.19	6.38	1.06
Reach B	70	4.87 (0.04-117)	37.1	18.6	22.9	8.57
Reach C	88	2.58 (0.03-56.7)	36.4	11.4	18.2	3.41
Reach D	75	1.97 (0.02-91.3)	32.0	4.00	10.7	1.33
Reach E	163	2.29 (0.02-45.4)	35.0	11.0	14.1	3.68
Reach F	88	1.09 (0.02-12.2)	39.8	10.2	15.9	0
Reach G	90	3.01 (0.03-51.9)	56.7	13.3	40.0	5.56
Reach H	136	3.97 (0.03-63.7)	82.4	16.9	52.9	5.15
Reach I	227	0.40 (0.03-5.91)	28.6	0.44	4.41	0

a. Mean (minimum – maximum).
b. 0.4 mg/kg.
c. 1.7 mg/kg.
Source: Blasland, Bouck & Lee, 2001.

4.4 Dietary Exposure to Mink

PCBs are not readily degraded and are persistent in the environment, and their concentrations can increase up a food chain once they enter a food chain. PCBs in the KRE that accumulate in fish can be consumed by piscivores such as mink. Fish comprise a large portion of the mink diet, although mink also consume muskrats, crayfish, small rodents, and birds (Camp Dresser & McKee, 2003b). Thus mink can be exposed to PCBs that are accumulated by fish from KRE sediment and surface water.

As part of the ecological risk assessment for the KRE site, EPA and the State of Michigan prepared a PCB food chain model (Camp Dresser & McKee, 2003b). The model incorporates a quantitative link between PCB concentrations in surficial sediments of the Kalamazoo River and PCB concentrations in fish, and thus allows for the prediction of PCB concentrations in fish based on PCB concentrations in surficial sediment in areas where the fish live and forage. Given a PCB concentration in fish that causes injury to mink that consume the fish, the model can thus be used to back-calculate the surficial sediment PCB concentration that corresponds to the mink injury. The model thus can be used to identify areas where PCB concentrations in surficial sediment are sufficient to cause injury to mink via the mink's consumption of fish exposed to the PCB contaminated sediment.

4.4.1 Data sources

The data used to evaluate injury to sediment based on mink dietary exposure are the same as were used to evaluate injury to sediments based on invertebrate SECs and are described in Section 4.3.1. As in Section 4.3, data are summarized by reaches. Foraging ranges for mink are consistent with the approximate lengths of these reaches (U.S. EPA, 1993), and thus it is reasonable to evaluate mink exposure to PCBs in river sediment on this basis.

4.4.2 Toxicity reference value derivation

The April 2003 MDEQ Baseline Ecological Risk Assessment (ERA) (Camp Dresser & McKee, 2003b) developed by the MDEQ for the Portage Creek/Kalamazoo River site derived site-specific sediment thresholds from a dietary exposure model for higher trophic level organisms that consume fish, including mink.

Mink were the most sensitive species identified in the ERA and were thus used to develop a sediment threshold concentration. The MDEQ developed no and low effect toxicity reference values (TRVs) for mink from exposure-response curves compiled from the results of numerous toxicity studies. The no effect TRV is an estimate of the highest dietary dose of PCBs that mink can consume without adverse effects occurring, and the low effect TRV is an estimate of the lowest dietary dose at which adverse effects occur. MDEQ derived a no effect TRV of 0.5 mg PCB/kg ww diet, based on the effects of Aroclor 1254 on the number of live kits per mated female and kit body weight. The low effect TRV of 0.6 mg PCB/kg ww diet developed by MDEQ was based on the effects of Aroclor 1254 on the number of live kits per mated female.

Using observed data for surface water and fish PCB concentrations, MDEQ calculated a site-specific bioaccumulation factor (BAF) of 305,000, which represents the observed ratio of PCB concentration in fish to PCB concentration in water in the KRE (Camp Dresser & McKee,

2003b). MDEQ assumed that the mink diet is comprised of 100% fish, and derived surface water thresholds corresponding with the no effect and low effect TRVs for mink using the equation:

$$\textit{surface water PCB threshold} = \frac{\textit{no effect or low effect TRV}}{\textit{site-specific BAF}}.$$

The corresponding sediment threshold was calculated using a site-specific sediment to surface water partition factor (K_d) of 302,000, which represents the observed ratio of PCB concentration in sediment to PCB concentration in water in the KRE (Camp Dresser & McKee, 2003b). Finally, a sediment threshold corresponding with the no effect TRV (0.5 mg/kg) and the low effect TRV (0.6 mg/kg) for mink was derived using the formula:

$$\textit{sediment PCB threshold} = \textit{surface water PCB threshold} * \textit{site-specific } K_d.$$

These concentrations (Table 4.5) represent the ERA's estimate of sediment PCB concentrations that would result in mink being exposed to PCBs in their diet at the no effect and low effect levels.

Table 4.5. Site-specific sediment thresholds for the protection of mink

Species	No effect TRV (mg PCB/kg sediment dw)	Low effect TRV (mg PCB/kg sediment dw)
Mink	0.5	0.6

Source: Camp Dresser & McKee, 2003b.

This Stage I injury assessment uses low effect TRVs or LOECs as injury thresholds. Although LOECs may overestimate true injury thresholds (i.e., underpredict toxicity and injury), the Trustees used the LOECs in this assessment because they represent dose or exposure concentrations where adverse effects have been shown to occur and therefore, provide a more certain estimate of injury than no effect concentrations, or NOECs. Therefore, the Trustees selected the mink low effect TRV of 0.6 mg/kg dw to evaluate injury to sediments.

4.4.3 Results

The site-specific TRV for mink is exceeded in all of the assessment reaches from Portage Creek and the city of Kalamazoo as far downstream as Lake Michigan (Figure 4.3). Across all of the assessment area, PCB concentrations in 31% of the surface sediment samples exceed the TRV for mink. In upstream reference reaches, 15% of samples have concentrations that are higher than the low effect TRV for mink.

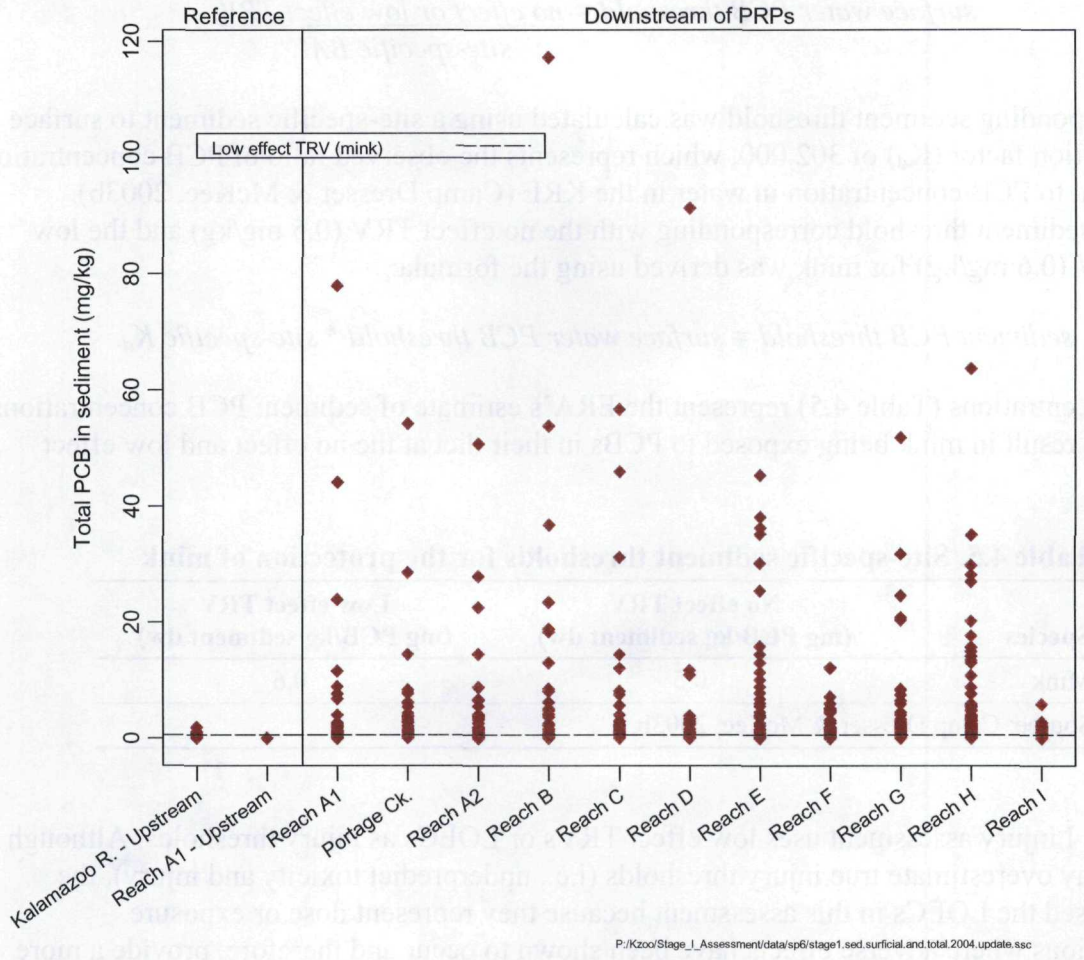

Figure 4.3. Depth-weighted mean PCB concentrations in surficial sediment from cores collected in Portage Creek and the Kalamazoo River compared to site-specific TRV for mink.

Sources: Blasland Bouck & Lee, 2001; Camp Dresser & McKee, 2003b.

Within individual assessment reaches, the percentage of surface samples which exceed the TRV for mink ranges from 15.1% in Reach A1 to 79.6% in Portage Creek (Table 4.6). In all assessment reaches except for Reach I, some samples have PCB concentrations greater than ten times the TRV for mink. These high PCB concentrations in surface sediment exceed the TRVs that the Trustees selected, and therefore, the Trustees believe that the data support the conclusion that mink (and, potentially, other biota with similar exposure and sensitivity to PCBs) are impaired by PCBs in sediments that enter the aquatic food chain. Because of the history of PCB releases from paper company facilities into the KRE, these adverse effects are likely to have begun decades ago.

Table 4.6. Exceedences of site-specific TRV for mink by depth-weighted mean PCB concentrations in surficial sediment of Portage Creek and the Kalamazoo River

Reach	Number of samples	PCB concentration (mg/kg)[a]	% exceeding TRV for mink[b]	% exceeding TRV for mink by > 10 times
Kalamazoo River – upstream	22	0.39 (0.03-1.88)	27.3	0
Reach A1 – upstream	17	0.03 (0.02-0.05)	0	0
Reach A1	166	1.33 (0.02-78)	15.1	4.22
Portage Creek	49	4.03 (0.07-54.3)	79.6	14.3
Reach A2	282	0.79 (0.02-50.8)	15.2	2.48
Reach B	70	4.87 (0.04-117)	31.4	14.3
Reach C	88	2.58 (0.03-56.7)	29.5	7.95
Reach D	75	1.97 (0.02-91.3)	28.0	4.00
Reach E	163	2.29 (0.02-45.4)	25.8	9.82
Reach F	88	1.09 (0.02-12.2)	34.1	3.41
Reach G	90	3.01 (0.03-51.9)	54.4	10.0
Reach H	136	3.97 (0.03-63.7)	77.9	11.8
Reach I	227	0.40 (0.03-5.91)	19.8	0

a. Mean (minimum – maximum).
b. 0.6 mg/kg.
Source: Blasland, Bouck & Lee, 2001.

4.5 Temporal Extent

This chapter evaluates injuries to sediments using PCB concentrations in surface sediments collected between 1993 and 2000. Information on surface sediment concentrations in the past can be gained from consideration of dated sediment cores (see Section 2.4.2). Analysis of dated sediment cores indicates that PCB concentrations are highest in sediment deposited in the 1960s, when PCB releases were also high (Blasland, Bouck & Lee, 1994c). Although PCB

concentrations have decreased after the periods of peak releases to the river, deposition of PCBs in sediments has been steady since approximately 1980 (Blasland, Bouck & Lee, 1994c). Thus, injuries to sediments have occurred since the time of the initial releases from PRP facilities, and were likely higher in the past than they are today.

4.6 Conclusions

The Trustees conclude that PCB concentrations in surface sediment of Portage Creek and the Kalamazoo River downstream of PRP facilities are sufficient to have caused injury for decades.

Concentrations are up to nearly three orders of magnitude greater than a consensus-based extreme effect concentration above which toxicity to benthic invertebrates is expected to occur. Additionally, PCB concentrations in surface sediment are sufficient to have caused injury to mink and bald eagles based on food chain dietary exposure. Concentrations are as high as three orders of magnitude greater than a site-specific low effect TRV for mink. Concentrations in samples from all assessment reaches in Portage Creek and in the Kalamazoo River from the city of Kalamazoo to Lake Michigan exceed the mink TRV. Based on the history of PCB releases from the facilities (see Chapter 2) and the history of PCB concentrations from sediment core data, it is likely that PCB concentrations in sediment have been sufficient to have caused and will continue to cause injuries until the sediment PCB-to-receptor pathway is broken. Thus, the Trustees conclude that sediment is and has been injured throughout the KRE according to the injury definition in 43 C.F.R. § 11.62(b)(1)(v).

5. Injuries to Wildlife Services: Fish Consumption Advisories

5.1 Injury Definitions

The DOI regulations include two different injury definitions related to the effects of chemical contamination on human use and consumption of fish. According to these definitions, "injury to a biological resource has resulted from the . . . release of a hazardous substance if concentration of the substance is sufficient to:"

- Exceed levels for which an appropriate State health agency has issued directives to limit or ban consumption of such organism [43 C.F.R. § 11.62 (f)(1)(iii)]

- Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 C.F.R. § 11.62 (f)(1)(ii)].

In this chapter, injuries to fishery resources are determined using both of these regulatory tests.

5.2 Stage I Injury Assessment Approach

Table 5.1 outlines the approaches taken in this chapter to assess injury to fish according to the definitions listed above. The history and basis of fish consumption advisories (FCAs) is presented. Concentrations of PCBs in edible portions of fish are compared to FCA trigger levels used by the State of Michigan, and to federal tolerance levels.

5.3 History of KRE Fish Consumption Advisories

5.3.1 Summary of fish consumption advisories

FCAs have been issued for multiple species in the Kalamazoo River since 1979 (Figure 5.1). The reaches of the Kalamazoo River designated by the Michigan Department of Community Health (MDCH) for FCAs are from the confluence of the Kalamazoo River and the Battle Creek River to Morrow Dam, from Morrow Dam to Lake Allegan Dam (including Portage Creek), and downstream of Lake Allegan Dam. Additionally, advisories have been issued for Lake Michigan since 1977, and although these advisories are not specific to the Kalamazoo River, they apply to

Table 5.1. Approaches to evaluate injury to human use and consumption of fish

Injury definition	Stage I injury assessment approach	Chapter section
Exceed levels for which an appropriate state health agency has issued directives to limit or ban consumption of such organism [43 C.F.R. § 11.62 (f)(1)(iii)].	Present history and basis of fish consumption advisories in the Kalamazoo River, Portage Creek, and Lake Michigan.	5.3
	Compare concentrations of PCBs in edible portions of fish from the Kalamazoo River and Portage Creek to trigger levels used by Michigan.	5.4
Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342 [43 C.F.R. § 11.62 (f)(1)(ii)].	Compare concentrations of PCBs in edible portions of fish from the Kalamazoo River and Portage Creek to federal tolerance levels.	5.5

migratory fish that move up into the Kalamazoo River.[1] The advisories are communicated to the public in annual fishing guides published by MDNR (MDNR, 1977, 1978a, 1979, 1980, 1981b, 1982, 1983, 1984a, 1985, 1986, 1987c, 1988, 1989, 1990a, 1991, 1992a, 1993a, 1994a, 1995-2001).

The Kalamazoo River was not included in any FCAs before 1979 (Figure 5.1). FCAs have applied to carp (*Cyprinus carpio carpio*) and suckers (*Catostomus commersoni*) downstream of Morrow Lake since 1979. Catfish (*Ictalurus punctatus*), largemouth bass (*Micropterus salmoides*), smallmouth bass (*M. dolomieui*), and other species not identified specifically have been under advisory downstream of Morrow Lake for most years since 1979. In addition, northern pike (*Esox lucius*) have been under advisory downstream of Lake Allegan Dam since 1985.

The Lake Michigan advisories also apply to tributaries into which migratory species enter (MDNR, 2001). Thus the advisory for Lake Michigan south of Frankfort applies to the Kalamazoo River downstream of Lake Allegan Dam (Figure 5.1). Brown trout (*Salmo trutta trutta*), chinook salmon (*Oncorhynchus tshawytscha*), coho salmon (*O. kisutch*), lake trout (*Salvelinus namaycush*), rainbow trout (*O. mykiss*), and whitefish (*Coregonus clupeaformis*) have been under the Lake Michigan advisory since the late 1970s or early 1980s. Other species such as rainbow smelt (*Osmerus mordax*), sturgeon (*Acipenser fulvescens*), walleye (*Stizostedion vitreum*), and yellow perch (*Perca flavescens*) have been under advisories since the mid- to late 1990s.

1. The Lake Michigan advisories are the result of pollutant loadings into the lake from many different sources. Based on measurements of PCB loadings from the Kalamazoo River into Lake Michigan (U.S. EPA, 2000), it is reasonable to assume that KRE PCBs have contributed to the PCB advisories in the lake. Therefore, the Lake Michigan advisories are included in this presentation and discussion of FCAs relevant to the KRE.

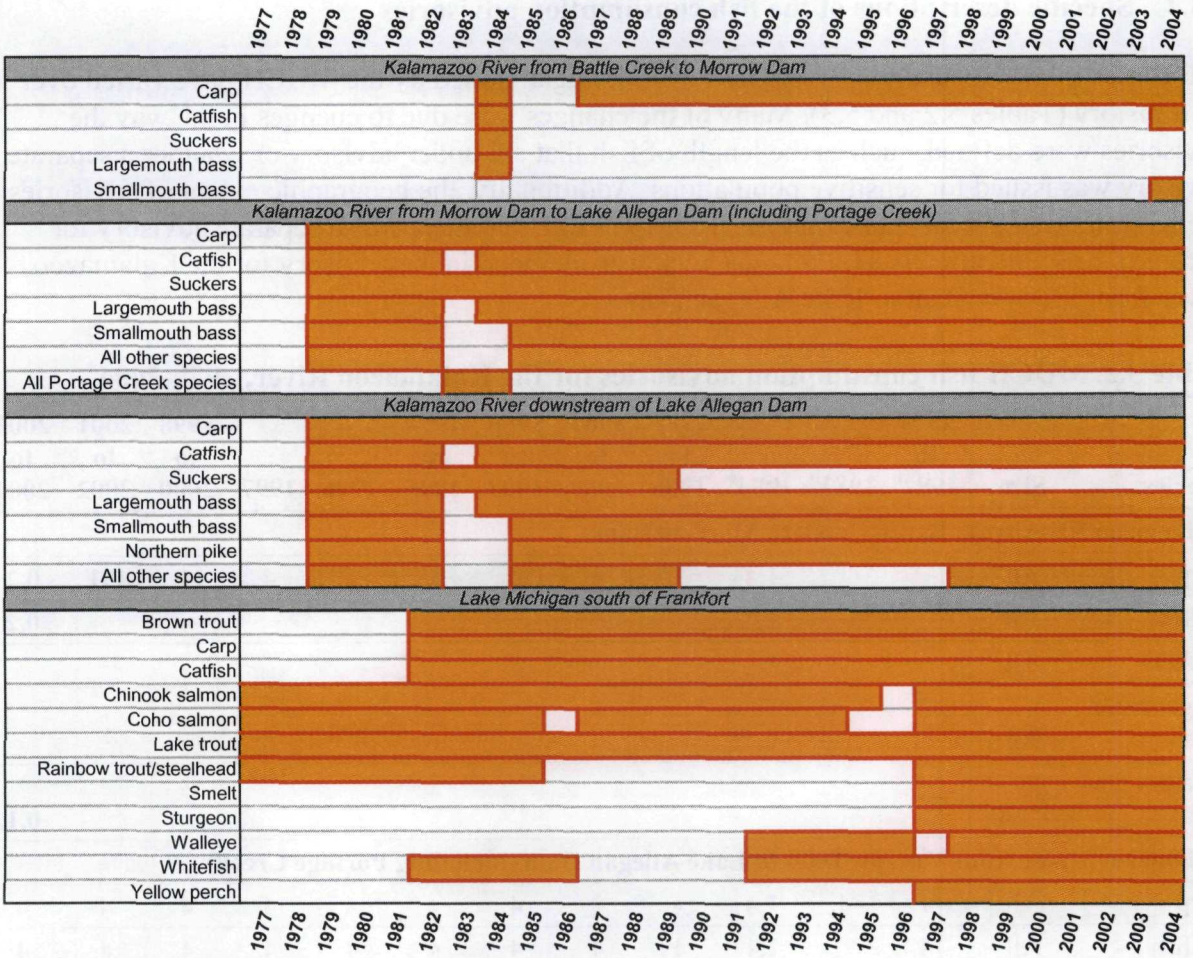

Figure 5.1. History of fish consumption advisories in the Kalamazoo River and Lake Michigan south of Frankfort. Orange bars indicate years in which there was an advisory of any kind.

Sources: MDNR, 1977, 1978a, 1979, 1980, 1981b, 1982, 1983, 1984a, 1985, 1986, 1987c, 1988, 1989, 1990a, 1991, 1992a, 1993a, 1994a, 1995-2001; U.S. EPA, 1997a; MDCH, 2002, 2003, 2004.

5.3.2 Specific descriptions of the fish consumption advisories

Advisories in the Kalamazoo River and Lake Michigan issued by the MDCH have varied over their history (Tables 5.2 and 5.3). Many of the changes were due to changes in the way the advisories were defined, such as the length of fish that are under advisory or whether a separate advisory was issued for sensitive populations. Additionally, the geographic extent of advisories varied from year to year. For example, from 1979 to 1983, there was a separate advisory for Portage Creek, but after 1983, Portage Creek was included in the advisory for the Kalamazoo River from Morrow Dam to Lake Allegan Dam.

Table 5.2. MDCH fish consumption advisories for the Kalamazoo River, 1979-2004[a]

Species	Size	1979 to 1982[b]	1983[b]	1984[c]	1985 to 1986	1987 to 1989	1990 to 1993	1994 to 1995	1996	1997	1998 to 2000	2001 to 2002	2003 to 2004
Kalamazoo River from Battle Creek to Morrow Dam													
Carp	All			1,4		4	4	4	4	4	4	0,1	0,2
Catfish	All			1,4									0,2
Suckers	All			1,4									
Largemouth bass	All			1,4									
Smallmouth bass	14″-30″												0,1
Kalamazoo River from Morrow Dam to Lake Allegan Dam (including Portage Creek)													
Carp	All	4	4	1,4	4	4	4	4	4	4	4	4	4
Catfish	All	4		1,4	4	4	4	4	4	4	4	4	4
Suckers	All	4	4	1,4	4	4	4	4	4	4	4	4	4
Largemouth bass	All	4		1,4	4	4	4	4	4				
	14″-30″									4	4	4	4
Smallmouth bass	All	4			1,4	1,4	4	4	4				
	14″-30″									4	4	4	4
All other species		4			1,4	1,4	1,4	1,4	1,4	1,4	1,4	1,4	1,4
All species in Portage Creek		4	4										

Table 5.2. MDCH fish consumption advisories for the Kalamazoo River, 1979-2004 (cont.)[a]

Species	Size	1979 to 1982[b]	1983[b]	1984[c]	1985 to 1986	1987 to 1989	1990 to 1993	1994 to 1995	1996	1997	1998 to 2000	2001 to 2002	2003 to 2004
Kalamazoo River downstream of Lake Allegan Dam													
Carp	All	4	4	1,4	4	4	4	4	4	4	4	4	4
Catfish	All	4		1,4	4	4	4	4	4	4	4	4	4
Suckers	All	4	4	1,4	4	4							
Largemouth bass	All	4		1,4	4	4		1,4	4				
	14″-30″									1,4	1,4	1,4	1,4
	> 15″						1,4						
Smallmouth bass	All	4			1,4	1,4		1,4	4				
	14″-30″									1,4	1,4	1,4	1,4
	> 15″						1,4						
Northern pike	All	4			1,4	1,4		4	4				
	≥ 22″									4	4	4	4
	20″-25″						1,4						
	> 25″						4						
All other species	All	4			1,4	1,4					0,2	0,2	0,2

4 = No consumption.
2 = Limit consumption to 1 meal (0.5 lb) per month.
1 = Limit consumption to 1 meal (0.5 lb) per week.
0 = Unlimited consumption.

a. If there is only one symbol it is the advice for the whole population. When two numbers are shown, the first is the advice for the "general population" and the second is the advice for "children and women who are pregnant, nursing, or expect to bear children." From 1979 to 1983 children are not defined by age, from 1984 to 1987 the advice is for children age 6 and under, and from 1988 to 2004 the advice is for children age 15 and under.

b. From 1979 to 1983 there is a separate advisory for "all other species" in Portage Creek; thereafter Portage Creek species are included in the Kalamazoo River from Morrow Dam to Lake Allegan Dam advisory.

c. In 1984, the advice was for the Kalamazoo River and Portage Creek, with no distinction as to the reach.

Sources: MDNR, 1977, 1978a, 1979, 1980, 1981b, 1982, 1983, 1984a, 1985, 1986, 1987c, 1988, 1989, 1990a, 1991, 1992a, 1993a, 1994a, 1995-2001; MDCH, 2002, 2003, 2004.

Table 5.3. MDCH fish consumption advisories for Lake Michigan, south of Frankfort, 1977-2004[a]

Species	Size	1977 to 1981	1982 to 1985	1986	1987 to 1991	1992 to 1994	1995	1996	1997	1998	1999 to 2000	2001 to 2004
Brown trout	All		1,4	4								
	10″-22″				1,4	1,4			0	0,2	0,2	0,2
	≥ 22″				1,4	1,4			4	4	4	4
	> 23″				4	4	4	4				
Carp	All		1,4	4	4	4	4	4	4	4	4	4
Catfish	All		1,4	1,4	4	4	4	4	4	4	4	4
Chinook salmon	All	1,4	1,4									
	10″-26″									0,2	0,2	0,2
	≥ 26″			1,4						0,2	0,3	0,3
	21″-32″				1,4	1,4						
	> 32″				4	4	1,4					
Coho salmon	All	1,4	1,4									
	10″-30″									0,2	0,2	0,2
	> 26″				1,4	1,4						
	≥ 30″									0,3	0,3	0,3
Lake trout	All	1,4	1,4									
	10″-18″								0	0,2	0,2	0,2
	18″-22″								1,4	1,2	1,2	1,2
	≥ 22″								4	4	4	4
	20″-23″				1,4	1,4	1,4	1,4				
	> 23″				4	4	4	4				
	≤ 25″			1,4								
	> 25″			4								
Rainbow trout/ steelhead	All	1,4	1,4									
	10″-18″									0,1	0,1	0,1
	≥ 18″									0,2	0,2	0,2
Smelt	6″-14″									0,1	0,1	0,1
Sturgeon	≥ 30″								4	4	4	4
Walleye	14″-18″									0,1	0,1	0,1
	18″-22″									0,2	0,2	0,2
	22″-26″					1,2	1,2	1,2		1,2	1,2	1,2
	≥ 26″					1,2	1,2	1,2		1,3	1,3	1,3

Table 5.3. MDCH fish consumption advisories for Lake Michigan, south of Frankfort, 1977-2004 (cont.)[a]

Species	Size	1977 to 1981	1982 to 1985	1986	1987 to 1991	1992 to 1994	1995	1996	1997	1998	1999 to 2000	2001 to 2004
Whitefish	All		1,4	1,4								
	6″-18″								0	0,1	0,2	0,2
	18″-22″								0	0,2	0,2	0,2
	≥ 22″								4	4	4	4
	> 23″					4	4	4				
Yellow perch	All											
	6″-8″									0,1	0	0
	8″-18″											0,1
	8″-22″									0,1	0,1	

4 = No consumption.
3 = Limit consumption to 6 meals (0.5 lb) per year.
2 = Limit consumption to 1 meal (0.5 lb) per month.
1 = Limit consumption to 1 meal (0.5 lb) per week.
0 = Unlimited consumption.

a. If there is only one symbol it is the advice for the whole population. When two numbers are shown, the first is the advice for the "general population" and the second is the advice for "children and women who are pregnant, nursing, or expect to bear children." From 1977 to 1983 children are not defined by age, from 1984 to 1987 the advice is for children age 6 and under, and from 1988 to 2004 the advice is for children age 15 and under.

Sources: MDNR, 1977, 1978a, 1979, 1980, 1981b, 1982, 1983, 1984a, 1985, 1986, 1987c, 1988, 1989, 1990a, 1991, 1992a, 1993a, 1994a, 1995-2001; MDCH, 2002, 2003, 2004.

In general, when advisories did not specify different levels of protection for general populations and sensitive populations, they were applied at the most restrictive level, "no consumption." From 1984 on, when separate advice was given for general and sensitive populations, advisories were less restrictive for the general population, but in most cases remained at the "no consumption" level for sensitive members of the population.

PCBs were the only contaminants identified as being responsible for the advisories in the Kalamazoo River and Portage Creek for 1979 to 1981 and for 1989 to 2004. From 1982 to 1988 the contaminant of concern was not identified by waterbody in the advisory; instead, a preamble said that the listed "locations contained one or more chemicals at levels of public health concern." However, since PCBs were identified in advisories prior to and after the 1982 to 1988 period when advisories did not identify specific contaminants, PCBs were also most likely responsible for the advisories in these years.

PCBs have also been identified as key contaminants for the Lake Michigan FCAs. Between 1977 and 1981, PCBs were one of the key contaminants listed for chinook salmon, coho salmon, lake trout, and rainbow trout. From 1982 to 1988, the contaminant of concern was not identified by waterbody or species in the Lake Michigan advisory; instead a preamble said that "locations contained one or more chemicals at levels of public health concern." PCBs have been listed as one of the contaminants responsible for advisories in all years since 1988, and therefore are also most likely responsible for the advisories from 1982 to 1988.

5.3.3 Regulatory criteria and standards

Until 1980, Michigan issued FCAs based on the U.S. Food and Drug Administration's (FDA's) tolerance level of 5 parts per million (ppm) PCB in edible tissue (trimmed and skinned fish; Humphrey and Hesse, 1986). In 1981, Michigan adopted a trigger level of 2 ppm PCB, three years before the level was adopted by the FDA, to reduce cancer risks.

The Lake Michigan states (Michigan, Indiana, Illinois, and Wisconsin) decided in 1987 to modify the application of the 2 ppm trigger level, so that advisories were based on the percentages of fish sampled that exceeded the trigger level (Hesse, 1997). The guidance for applying these trigger levels specifies that a more severe type of advisory should be used for a greater frequency of exceedence of a trigger level.

An effort to unify protocols among Great Lakes states was made in 1993. The Great Lakes Fish Consumption Advisory Task Force recommended a health protection value of 0.05 µg PCB/kg human body weight (BW)/day (Great Lakes Sport Fish Advisory Task Force, 1993). This value was determined by consensus and based on suspected risk to human health from available data. Based on this value, five recommended advisory levels were developed depending on PCB concentrations in edible fish tissue, assumed body weights, assumed fish weights, and a factor for reduction of PCBs through cleaning of fish (Table 5.4).

Table 5.4. 1993 advisory levels recommended by the Great Lakes Fish Consumption Advisory Task Force

Fish tissue PCB concentration (ppm)	Consumption advice
0.0-0.05	Unlimited consumption
0.06-0.2	One meal/week
0.21-1.0	One meal/month
1.1-1.9	Six meals/year
> 1.9	No consumption

Source: Great Lakes Sport Fish Advisory Task Force, 1993.

The Michigan Environmental Science Board reviewed these recommendations and developed recommendations in response (Fischer et al., 1995). This panel determined that risks were highest to children and women of child-bearing age (sensitive populations). To take into account the positive benefits of eating fish, the State of Michigan decided that the recommended advisory levels were appropriate for sensitive populations, but too restrictive for the general population. Thus, as of 1998, FCAs for the general population continued to be determined based on the 2 ppm trigger level, but FCAs for sensitive populations were determined based on the guidelines recommended by the Great Lakes Fish Consumption Advisory Task Force (Table 5.4). Trigger levels for the general and sensitive populations over time are outlined in Table 5.5.

Table 5.5. FCA trigger levels used by the State of Michigan since 1977

Time period	Trigger for general population FCA (ppm)	Trigger for sensitive population FCA (ppm)
1977-1980	5	5
1981-1997	2	2
1998-present	2	0.05

Source: Hesse, 1997.

The Michigan FCA process generally takes a conservative approach toward protection of the public health. The guidance document specifies that "prudent public health professional judgment" must be part of the decision making process (Humphrey and Hesse, 1986). For example, a precautionary position may be taken before data are available to fully characterize the degree of contamination. On the other hand, to remove an advisory, "adequate data showing the absence of chemical concentrations of concern must be available . . ." (Humphrey and Hesse, 1986). Therefore, concentrations exceeding the trigger levels are to be viewed as indicative of conditions that would prompt an advisory, but an advisory may be issued without extensive concentrations over a trigger level.

5.3.4 EPA supplementary fish consumption advisories

In 1997, EPA issued a FCA based on PCBs for Michigan's Great Lakes waters that was designed to supplement the existing advisory from the MDCH (Table 5.6; U.S. EPA, 1997a). This advisory was more restrictive for certain species than those advisories developed by MDCH. For example, although MDCH advised unlimited consumption of brown trout from 10 to 22 inches in 1997, the EPA advised that the general population limit consumption to one meal per month. The advisories for lake trout and whitefish were also more restrictive, recommending restricted consumption of smaller fish when the MDCH did not. The EPA advisory also included several species that were not under advisories by the MDCH in 1997, such as coho salmon, chinook salmon, rainbow trout, yellow perch, and smelt.

Table 5.6. Comparison of MDCH and EPA fish consumption advisories for Lake Michigan south of Frankfort, 1997

Species	Size	State of Michigan advisory	EPA supplemental advisory[a]
Coho salmon	All		2
Chinook salmon	10″-30″		2
	> 30″		3
Rainbow trout	10″-18″		1
	> 18″		2
Brown trout	10″-22″	0	2
	> 22″	4	4
Yellow perch	6″-22″		1
Smelt	6″-14″		1
Lake trout	10″-18″	0	2
	18″-22″	1,4	2,4
	22″- > 30″	4	4
Lake whitefish	6″-18″	0	1
	18″-22″	0	2
	22″- > 30″	4	4

4 = No consumption.
3 = Limit consumption to 6 meals (0.5 lb) per year.
2 = Limit consumption to 1 meal (0.5 lb) per month.
1 = Limit consumption to 1 meal (0.5 lb) per week.
0 = Unlimited consumption.
a. If there is only one symbol it is the advice for the whole population. When two numbers are shown, the first is the advice for the "general population" and the second is the advice for "women of childbearing age and children."
Sources: MDNR, 1997; U.S. EPA, 1997a.

Although the supplementary EPA advisory may not satisfy the injury definition in the DOI regulations at 43 C.F.R. § 11.62 (f)(1)(iii) because it was issued by EPA and not a state agency, the advisory nevertheless was issued by EPA out of public health concerns over human consumption of PCB contaminated fish (Section 5.4.2 presents a description of the basis for the supplementary advisories). Furthermore, the advisory was issued to the public and, to the extent that it affected public use or values of recreational fishing, the advisory is relevant to the discussion of recreational fishing damages (see Michigan Department of Environmental Quality et al., 2005).

5.4 Exceedences of the Michigan FCA Trigger Levels

This section compares PCB concentrations measured in KRE fish tissue with the FCA trigger levels that have been used by the MDCH [43 C.F.R. § 11.62 (f)(1)(iii)].

5.4.1 Data sources

General guidelines for analysis of fish samples for consumption advisories are to sample only edible portions (fillets). Fillet PCB concentration data are available from several data sources:

- Characterization studies conducted primarily by the MDNR (MWRC, 1972a; Wuycheck, 1978; Horvath and Greminger, 1982; MDNR, 1984b, 1987b, 1992b, 1994b; State of Michigan, 1987; Blasland, Bouck & Lee, 1992)
- Studies conducted in 1993, 1997, and 1999 by Blasland, Bouck & Lee (2001) for the RI/FS
- Long-term monitoring studies conducted in 1999 and 2000 by Camp Dresser & McKee (2001, 2002b) on behalf of MDEQ.

Characterization studies were conducted primarily by the MDNR from 1971 to 1993. The Trustees compiled the results of these studies into a database, using primary sources where available (MWRC, 1972a; Wuycheck, 1978; Horvath and Greminger, 1982; MDNR, 1984b, 1987b, 1994b; State of Michigan, 1987), and supplementing these with additional data collected by various sources available in a compilation by Blasland, Bouck & Lee (1992) and data inventoried in the STORET database system (MDNR, 1992b). Species collected include bass (unidentified species), black crappie (*Pomoxis nigromaculatus*), bluegill (*Lepomis macrochirus*), bowfin (*Amia calva*), bullhead (*Ameiurus* sp.), carp, catfish, freshwater drum (*Aplodinotus grunniens*), largemouth bass, northern pike, pumpkinseed (*Lepomis megalotis*), rock bass (*Ambloplites rupestris*), smallmouth bass, trout, walleye, white sucker (*Catostomus commersoni*), and yellow perch. Fillet samples were collected as either individual skin-on fillets or individual skin-off fillets, and were analyzed for total PCBs.

Additional individual fish fillet PCB concentration data are from samples collected for the RI/FS by Blasland, Bouck & Lee in 1993, 1997, and 1999 (Blasland, Bouck & Lee, 2001). Fish were collected by electroshocking at 12 "Aquatic Biota Sampling Areas" (ABSAs) in 1993 (Blasland, Bouck & Lee, 1994b, 1994d). Carp and smallmouth bass fillets were collected at a subset of locations in 1997 (Blasland, Bouck & Lee, 2000b). Additional fillet samples were collected in 1999 at nine of the original ABSAs, and at a new site near Saugatuck (Blasland, Bouck & Lee, 2000c). Fish collected in 1999 included black crappie, bluegill, carp, catfish, northern pike,

pumpkinseed, smallmouth bass, and walleye. Carp, catfish, and northern pike were prepared for analysis as skin-off fillets, and smallmouth bass and all other species were prepared as skin-on fillets. All samples were analyzed for PCBs as Aroclors.

Individual fish fillet PCB concentration data are also taken from samples collected as a part of long-term monitoring by Camp Dresser & McKee (2001, 2002b) on behalf of MDEQ. In 1999, carp and smallmouth bass were collected at 10 locations between Ceresco and Kalamazoo Lake by electrofishing. Additionally, one brown trout was collected from Kalamazoo Lake, two channel catfish were collected from Lake Allegan and New Richmond, and two largemouth bass were collected from New Richmond. In 2000, carp and smallmouth bass were collected from Portage Creek and four locations along the Kalamazoo River mainstem from Ceresco to Lake Allegan. Additionally, one brown trout was collected from Portage Creek, one northern pike and one rock bass were collected in the city of Kalamazoo, and one black crappie, one largemouth bass, and two walleye were collected from Lake Allegan.

All concentrations presented in this chapter are expressed on a wet weight basis. Parts per million is used to express concentrations in tissue for consistency with most of the fish consumption advisory literature, which uses ppm rather than mg/kg to describe concentrations in fish. Data used for this evaluation are as presented in source documents. No corrections or efforts to normalize the data were made to account for variability in fish length, lipid concentration, or other factors.

All fillet PCB concentration data were grouped by the Trustees to correspond with the fish consumption advisory reaches based on either descriptive sample locations or ABSAs. The ABSAs correspond to the MDCH advisory reaches as follows (Figure 5.2):

- Kalamazoo River from Battle Creek to Morrow Dam: ABSA 1 (near Battle Creek) and ABSA 2 (Morrow Lake)

- Kalamazoo River from Morrow Dam to Lake Allegan Dam: ABSA 3 (upstream of Portage Creek), ABSA 12 (Portage Creek, former Bryant Mill Pond), ABSA 4 (near Mosel Ave. in Kalamazoo), ABSA 5 (upstream of Plainwell Dam), ABSA 6 (Plainwell Dam to Otsego City Dam), ABSA 7 (upstream of Otsego Dam), ABSA 8 (upstream of Trowbridge Dam), and ABSA 9 (Lake Allegan)

- Kalamazoo River downstream of Lake Allegan Dam: ABSA 10 (Swan Creek Marsh), ABSA 11 (near New Richmond), and ABSA 13 (near Saugatuck).

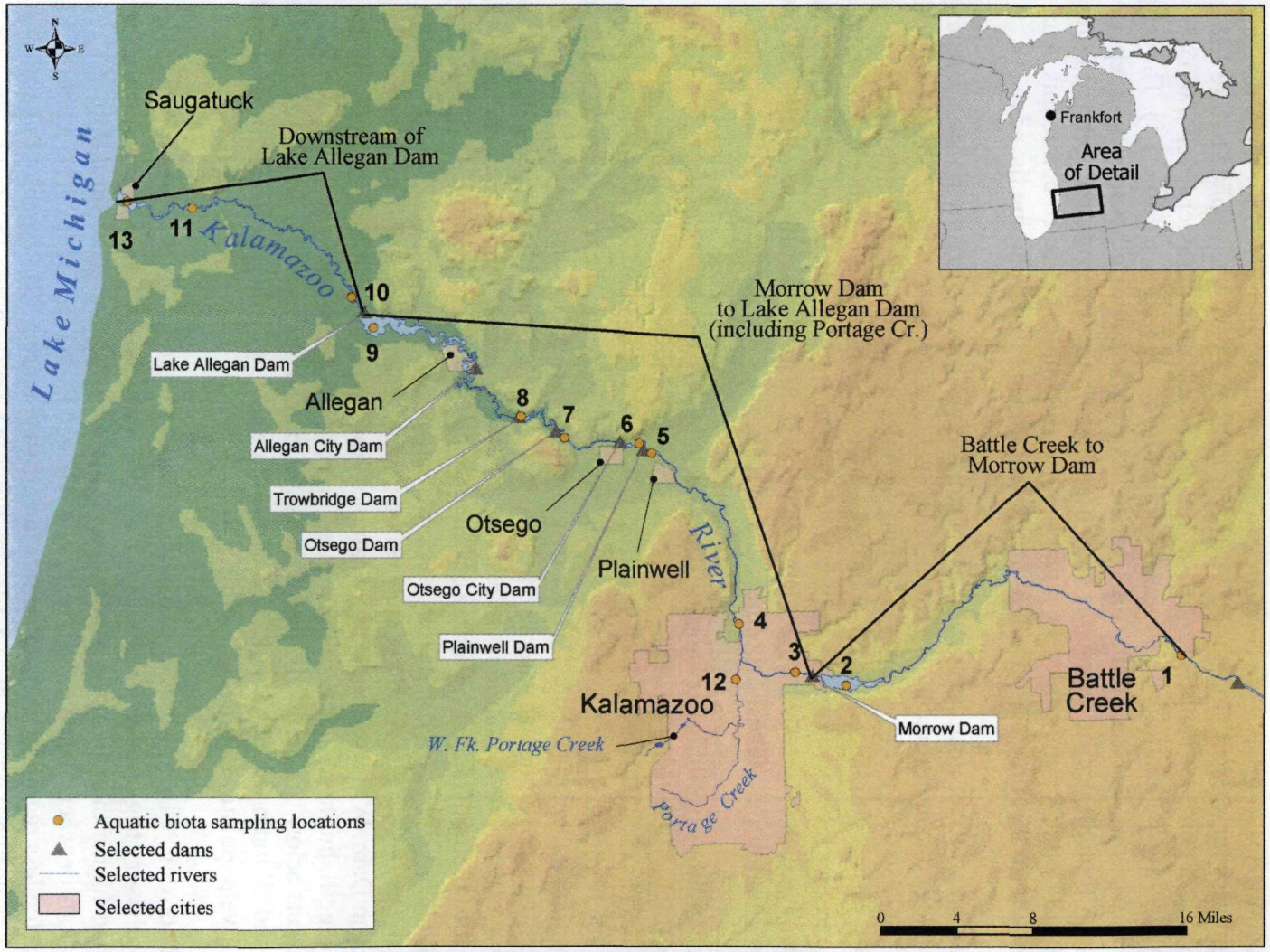

Figure 5.2. Reaches of the Kalamazoo River as designated by MDCH for fish consumption advisories, and distribution of RI/FS ABSAs within these reaches.

5.4.2 Results

PCB concentrations in fillets were compared to appropriate trigger levels for the Michigan advisory process for a given time period. A trigger level of 5 ppm was applied before 1981, a trigger level of 2 ppm was applied from 1981 on, and an additional trigger level of 0.05 for the minimum sensitive population advisory was applied from 1998 on (see Table 5.5). The purpose of these comparisons was to examine the consistency of Michigan's advisories with reported fish tissue data. Although specific advisories were not issued in the Kalamazoo River and Portage Creek until 1979, the 5 ppm trigger level was applied to samples collected before this year because data from this time period would have been relevant to developing the advisory.

In the Kalamazoo River upstream of Morrow Dam, only carp are currently under a consumption advisory. PCB concentrations in carp fillets have been measured at concentrations greater than the trigger levels since 1971 (Figure 5.3). Many samples exceeded the 2 ppm trigger level in the early 1980s, but no samples collected since 1987 have exceeded 2 ppm. More recent samples exceed the trigger level for sensitive populations. Although PCB concentrations in other species in this reach have also exceeded trigger levels on occasion, they generally have fallen below the 5 ppm and 2 ppm trigger levels over time (Figure 5.4).

PCB concentrations in edible portions of fish collected from Morrow Dam to Lake Allegan Dam were much higher than those upstream (Figures 5.5-5.10) and correspond with the more extensive advisories in this reach (see Table 5.2). Carp fillets had concentrations at least an order of magnitude higher than the trigger levels, and samples exceeded the trigger levels in all years when they were collected (Figure 5.5). Catfish samples were only collected in this reach in 1981 and 1999 (Figure 5.6). The one sample collected in 1981 fell slightly below the 2 ppm trigger level, however many of the samples collected in 1999 were higher than 2 ppm, and all of them exceeded the 0.05 ppm trigger level for sensitive populations. Largemouth bass (Figure 5.7), smallmouth bass (Figure 5.8), and white sucker (Figure 5.9) fillet samples also frequently exceeded the trigger levels. All other species are under a general advisory from Morrow Dam to Lake Allegan Dam. While the majority of samples from bass (unidentified species), black crappie, bluegill, bullhead, northern pike, pumpkinseed, rock bass, trout, and walleye had concentrations below trigger levels, many fillets did exceed the general population trigger level of 2 ppm and all but one bluegill fillet had concentrations higher than the 0.05 ppm trigger level for a sensitive population advisory (Figure 5.10).

Concentrations of PCBs in fillets of fish collected downstream of Lake Allegan Dam also exceeded advisory trigger levels (Figures 5.11-5.20). Many concentrations in carp fillets were several times higher than all of the trigger levels (Figure 5.11), and were similar to concentrations between Morrow Dam and Lake Allegan Dam (see Figure 5.5). Although samples were collected less regularly, concentrations in catfish (Figure 5.12), largemouth bass (Figure 5.13), northern pike (Figure 5.14), smallmouth bass (Figure 5.15), trout (Figure 5.16),

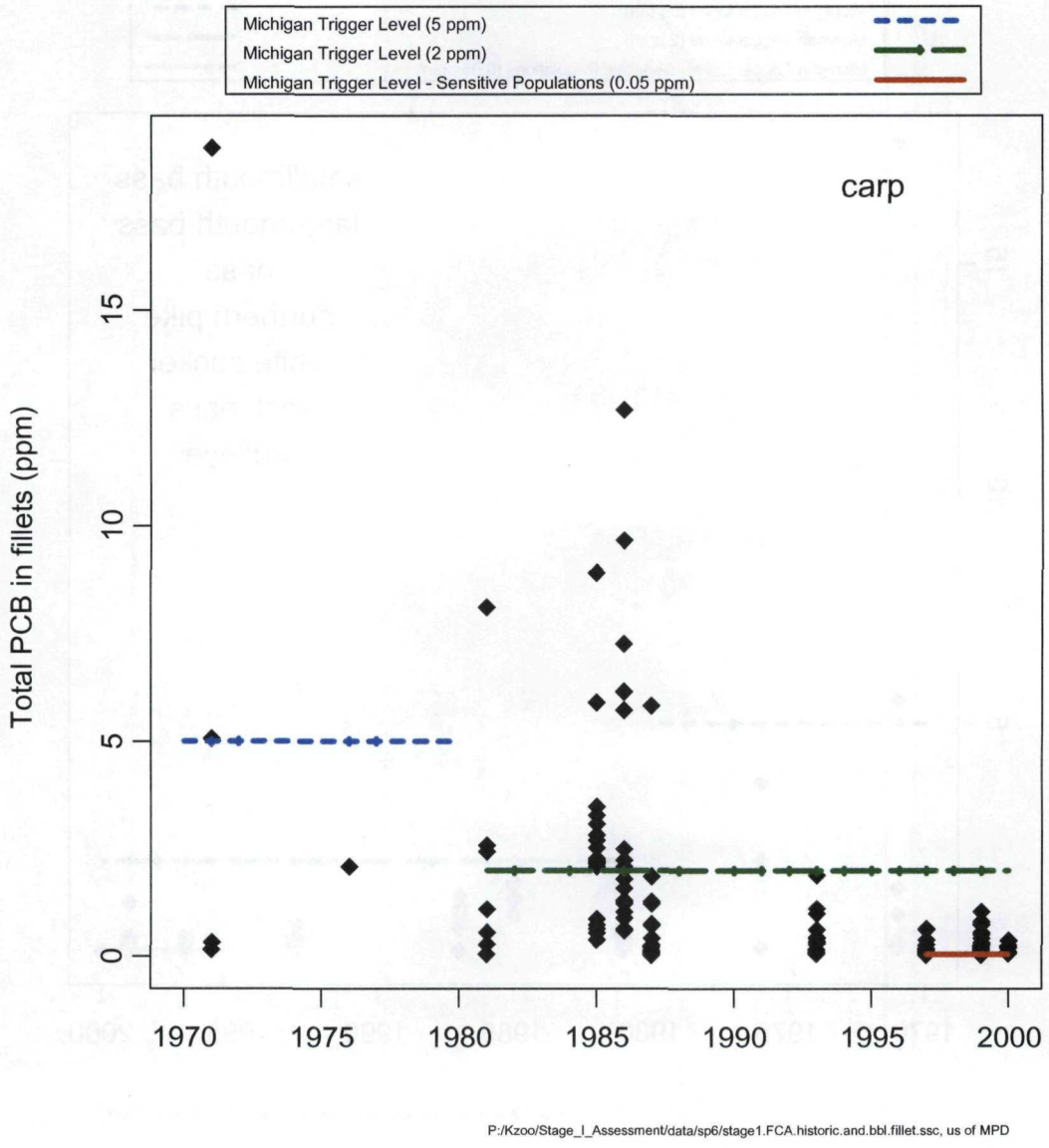

Figure 5.3. Total PCBs in fillets of carp collected upstream of Morrow Dam, 1971-2000.

Source: See Section 5.4.1.

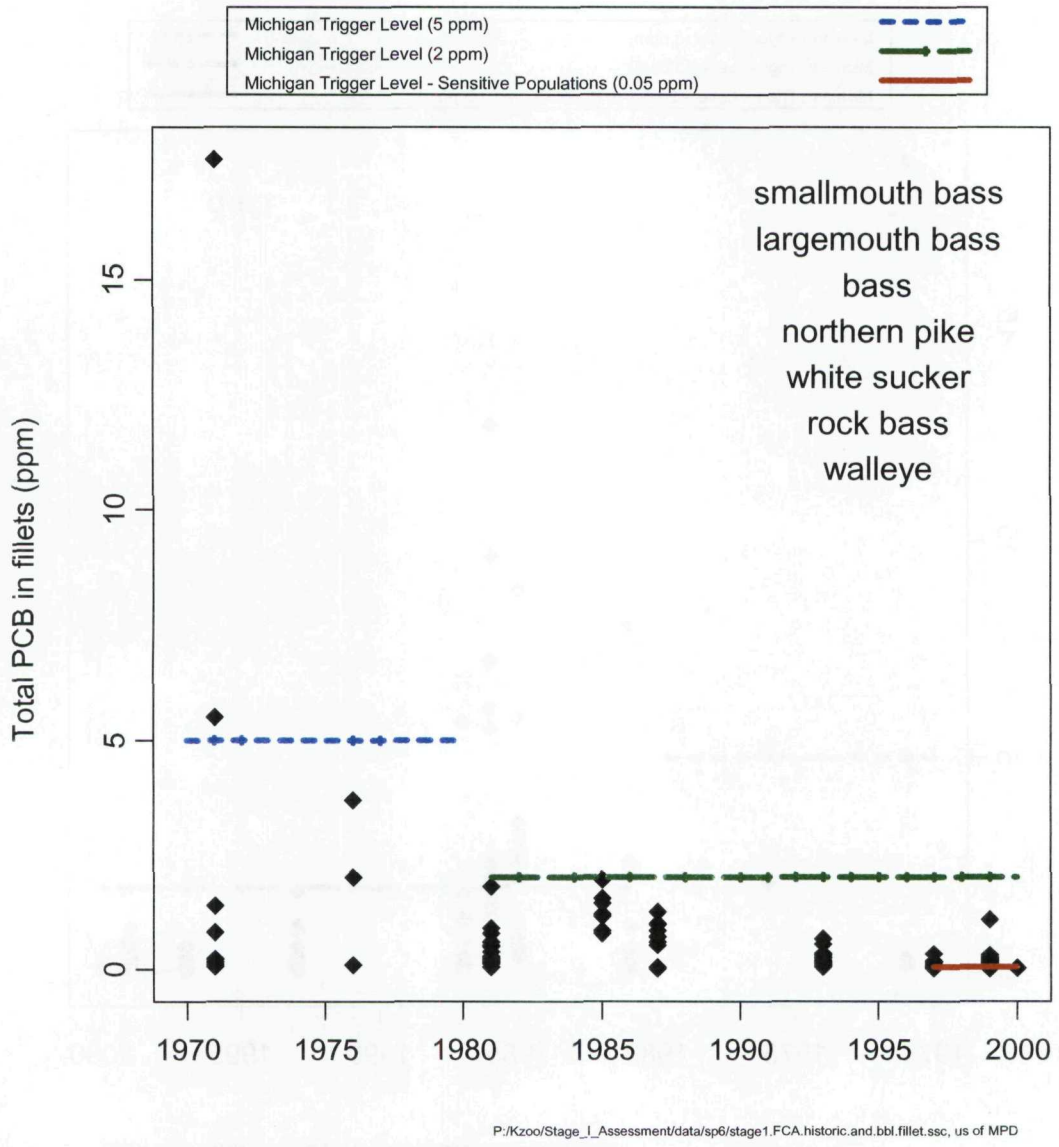

Figure 5.4. Total PCBs in fillets of species not currently under an advisory collected upstream of Morrow Dam, 1971-2000.

Source: See Section 5.4.1.

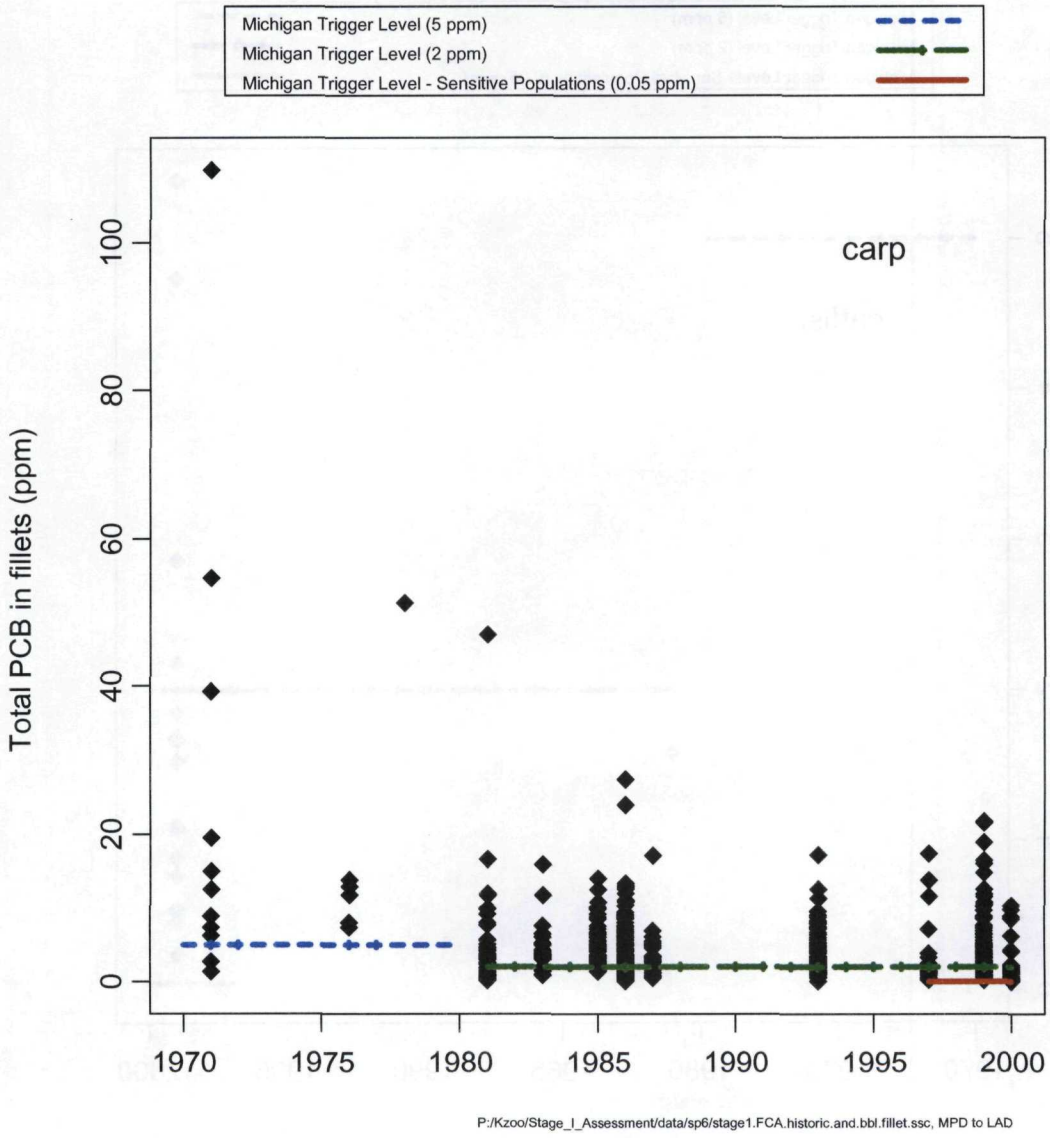

Figure 5.5. Total PCBs in fillets of carp collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

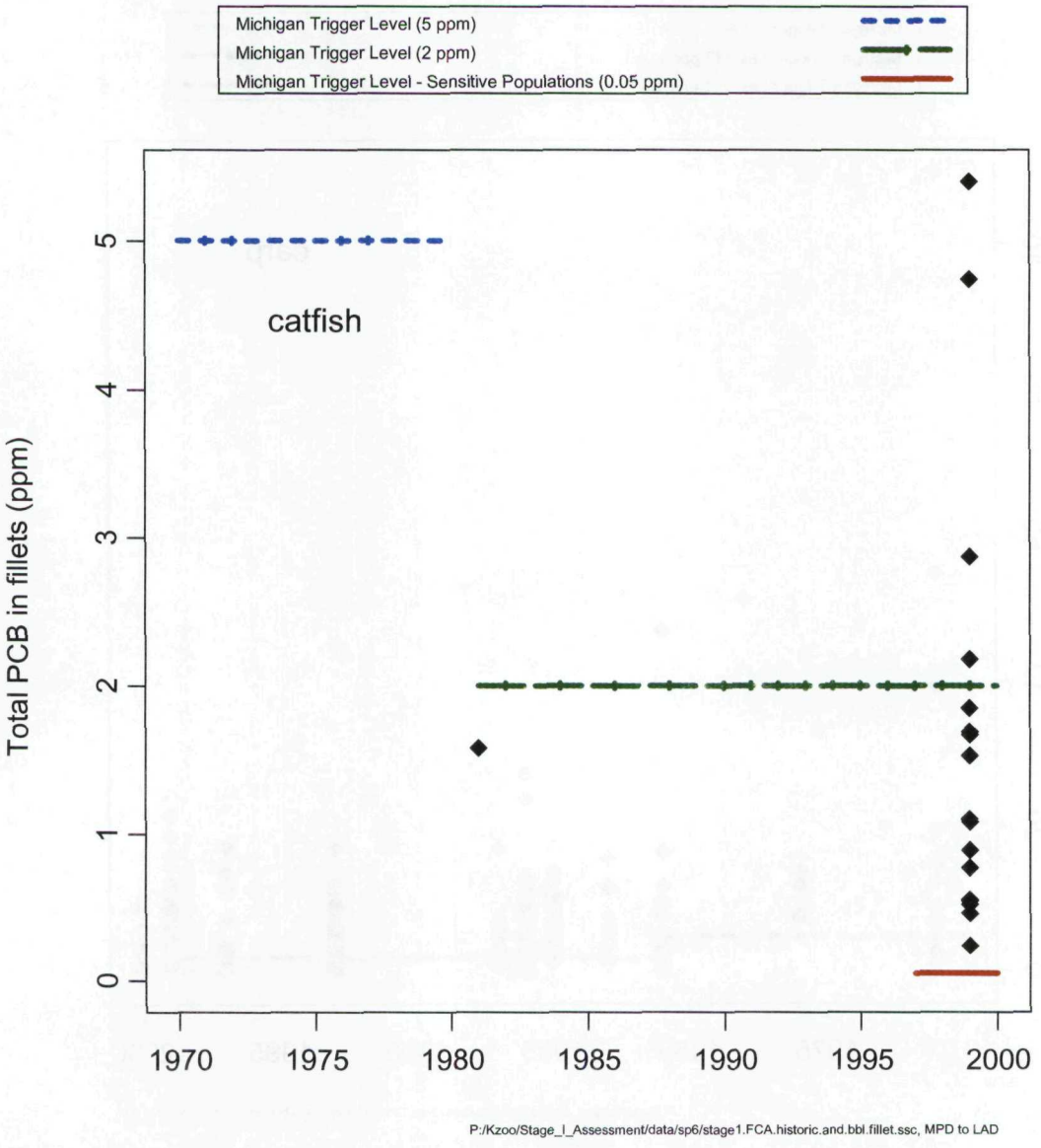

Figure 5.6. Total PCBs in fillets of catfish collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

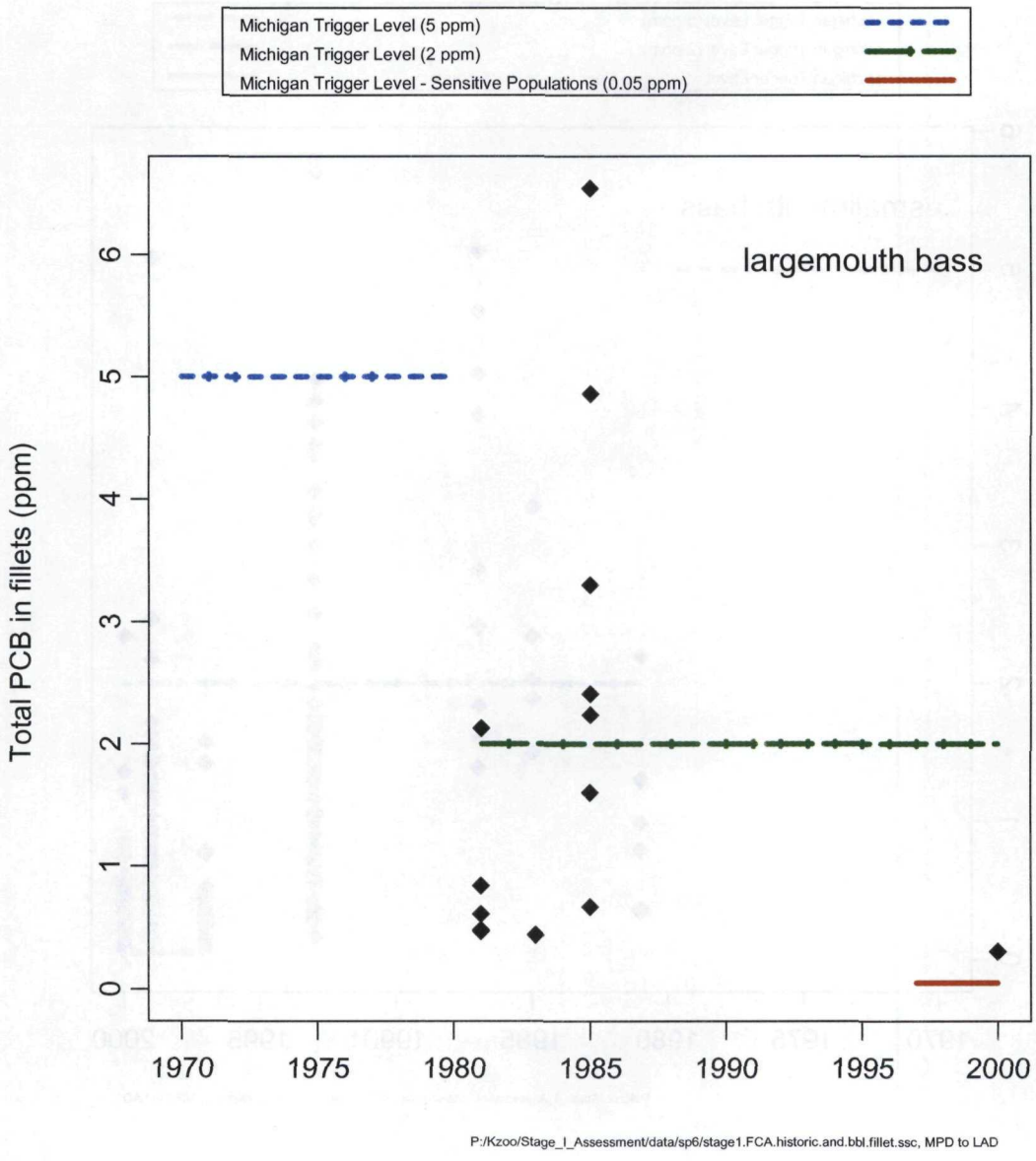

Figure 5.7. Total PCBs in fillets of largemouth bass collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

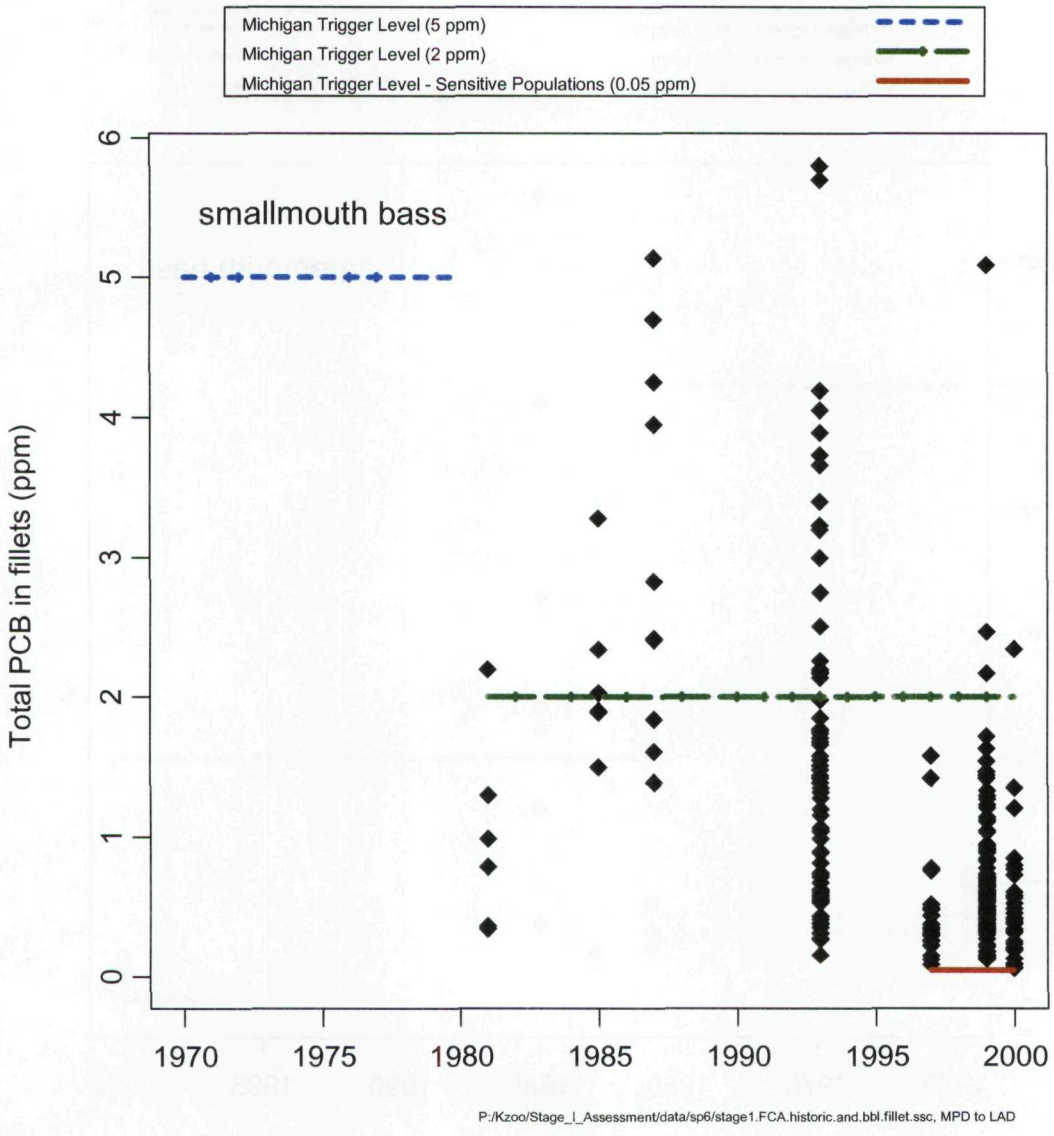

Figure 5.8. Total PCBs in fillets of smallmouth bass collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

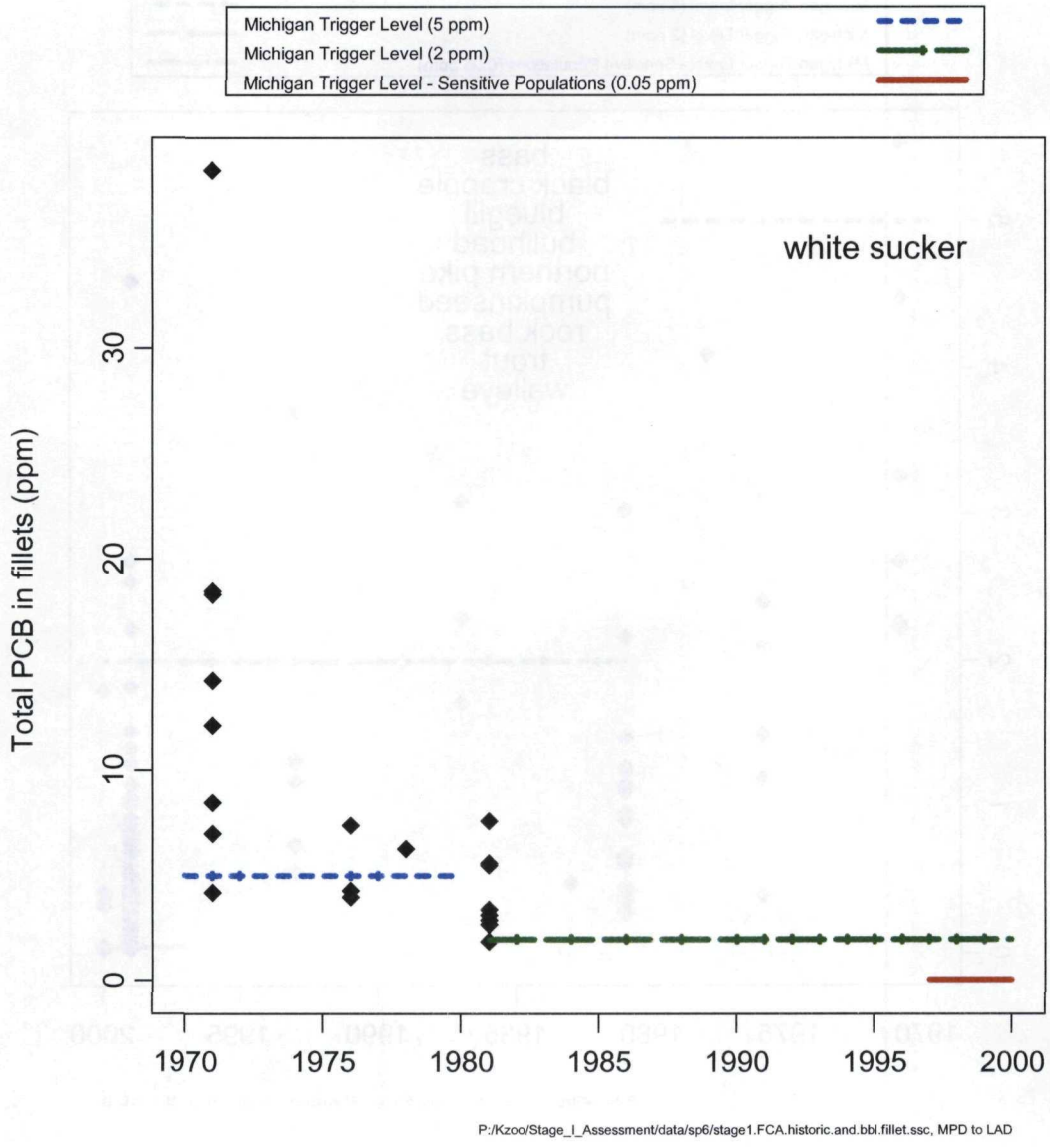

Figure 5.9. Total PCBs in fillets of white sucker collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

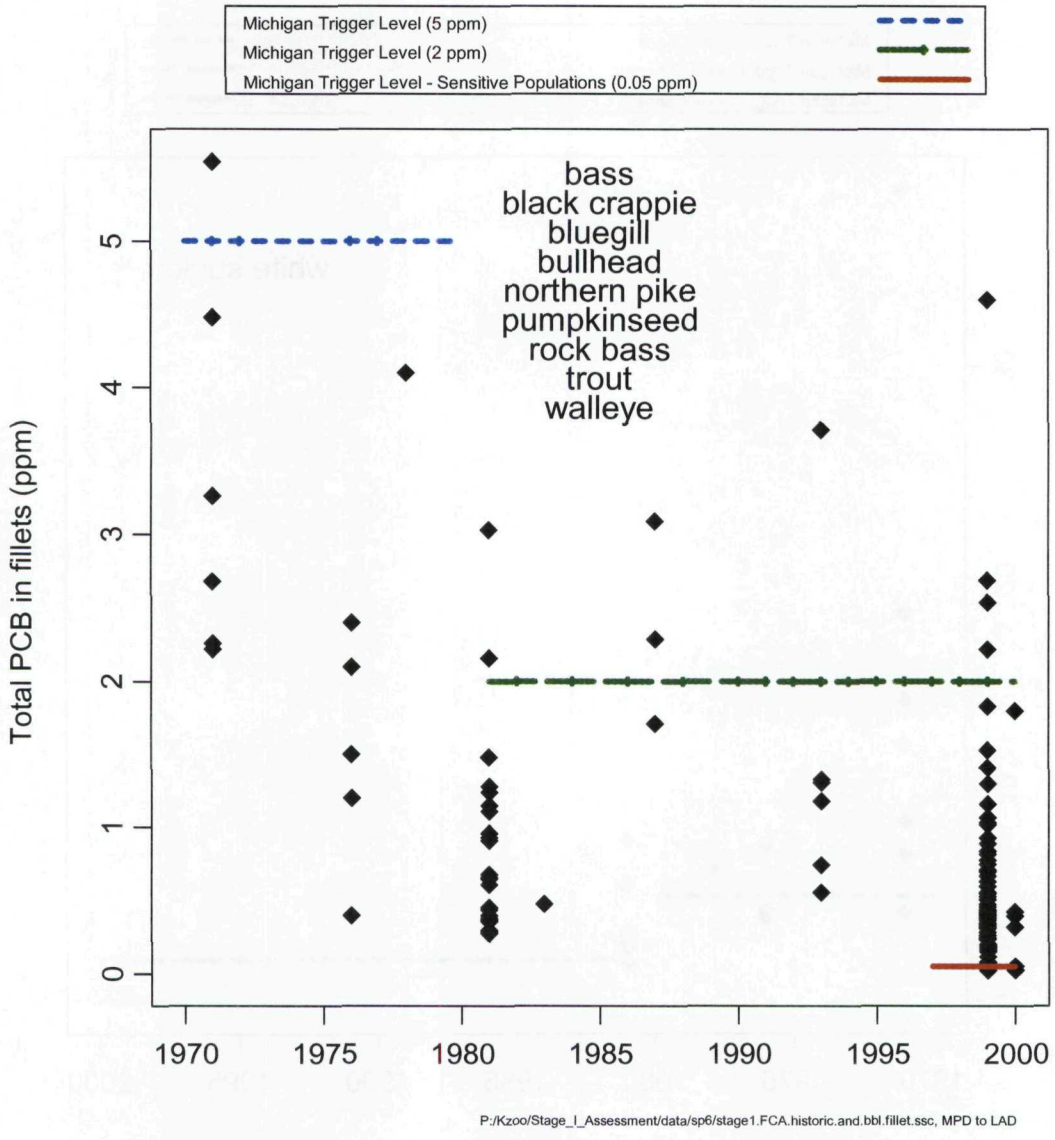

Figure 5.10. Total PCBs in fillets of other species under a general "all other species" advisory collected between Morrow Dam and Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

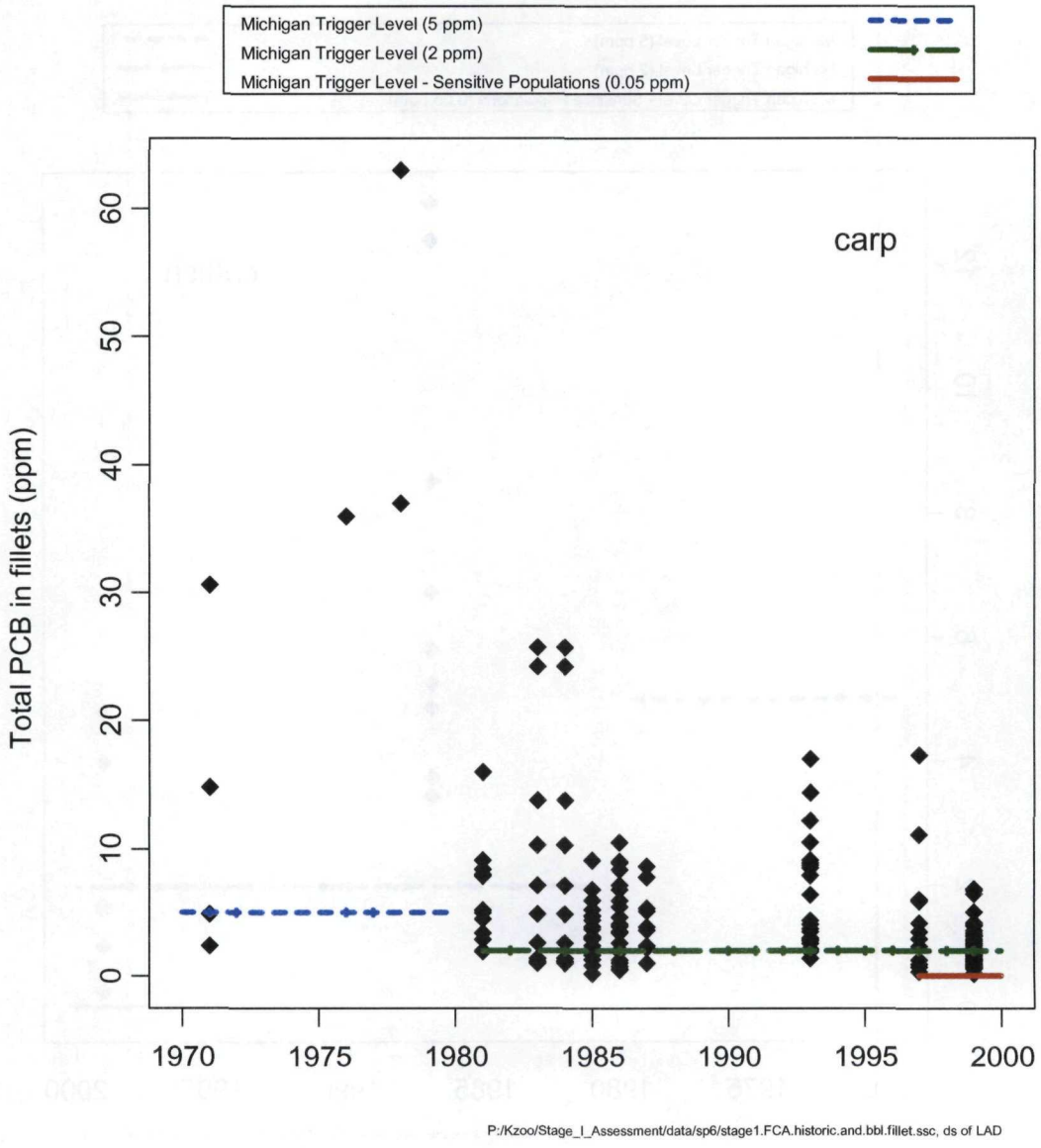

Figure 5.11. Total PCBs in fillets of carp collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

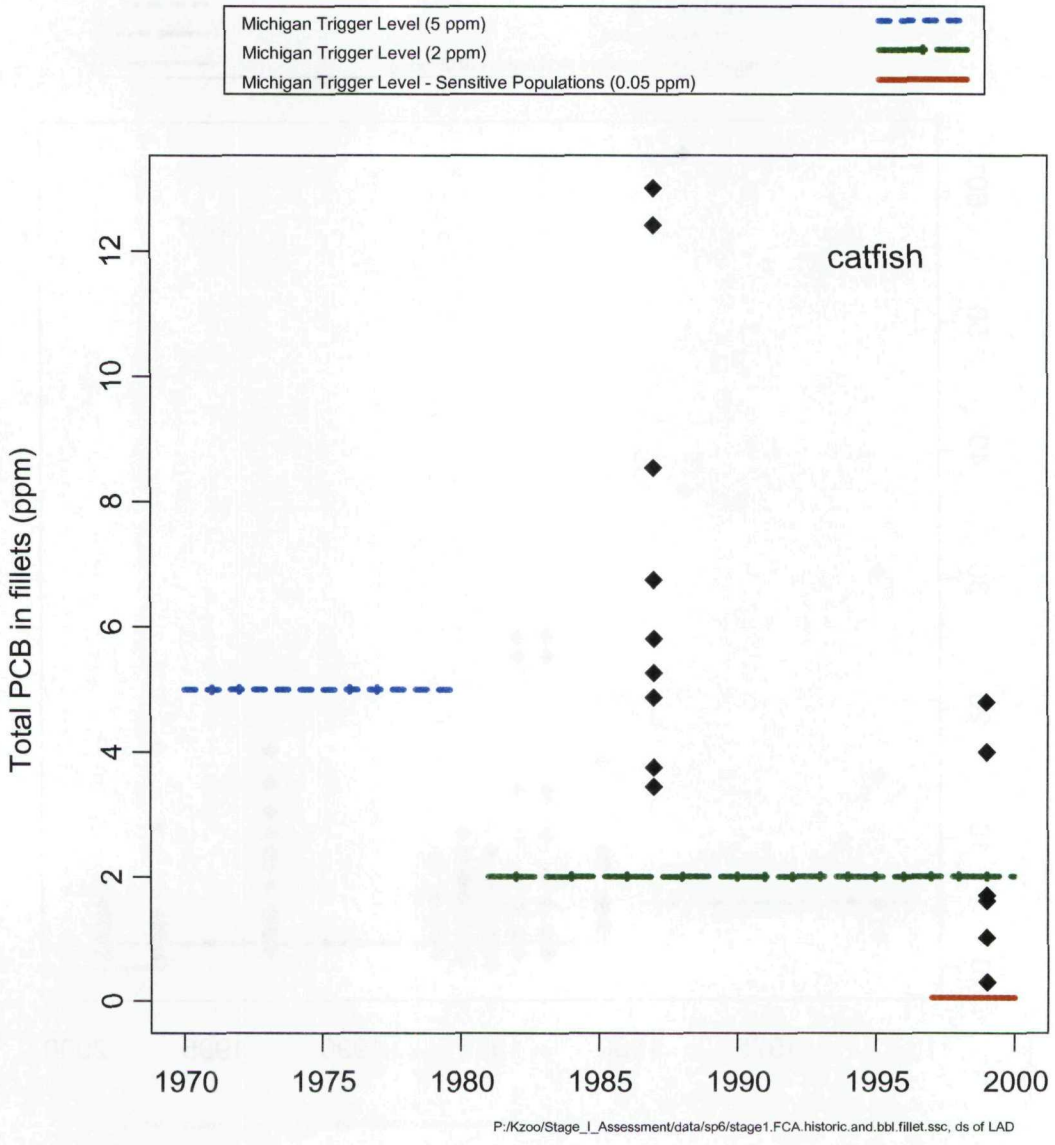

Figure 5.12. Total PCBs in fillets of catfish collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

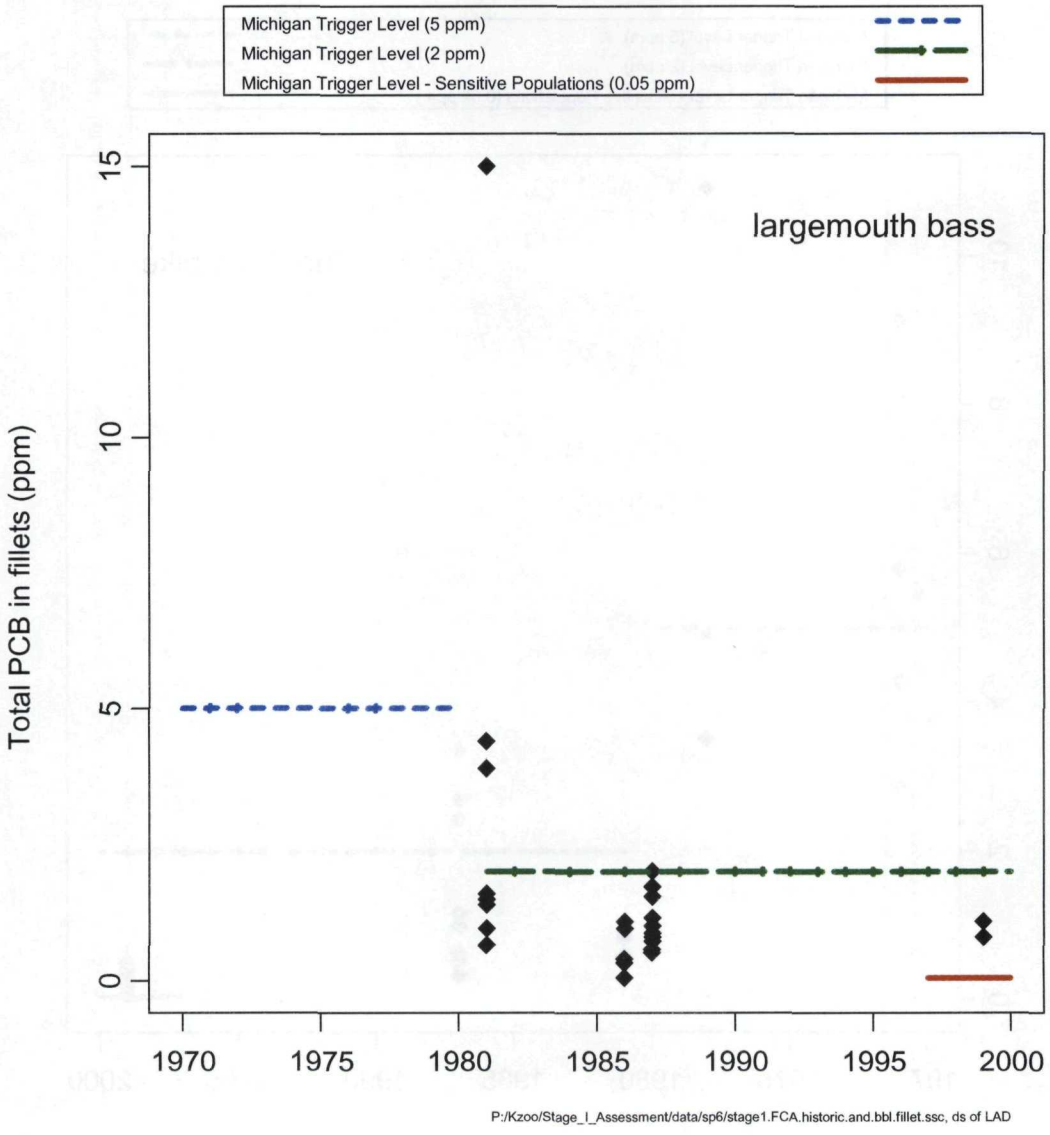

Figure 5.13. Total PCBs in fillets of largemouth bass collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

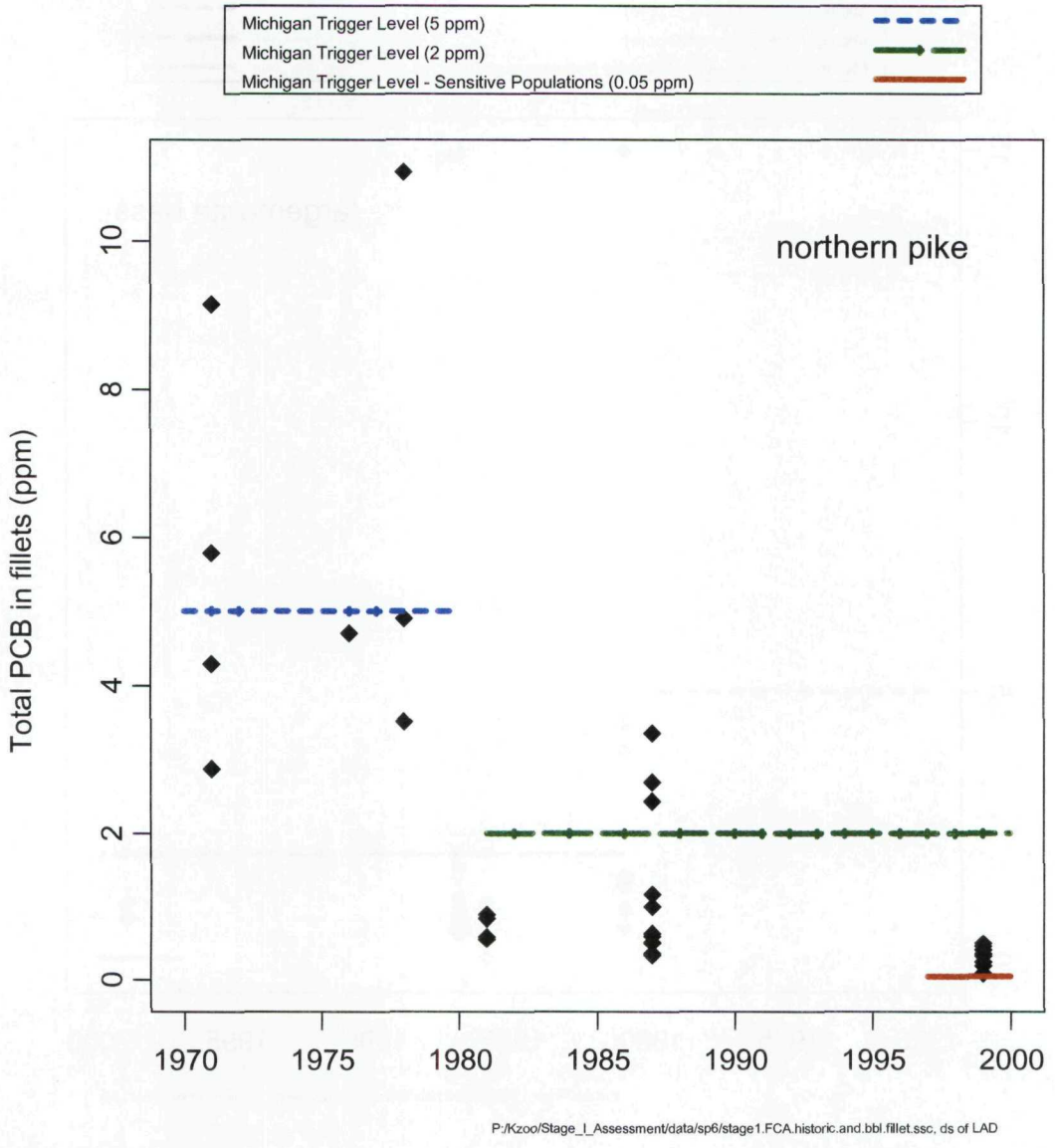

Figure 5.14. Total PCBs in fillets of northern pike collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

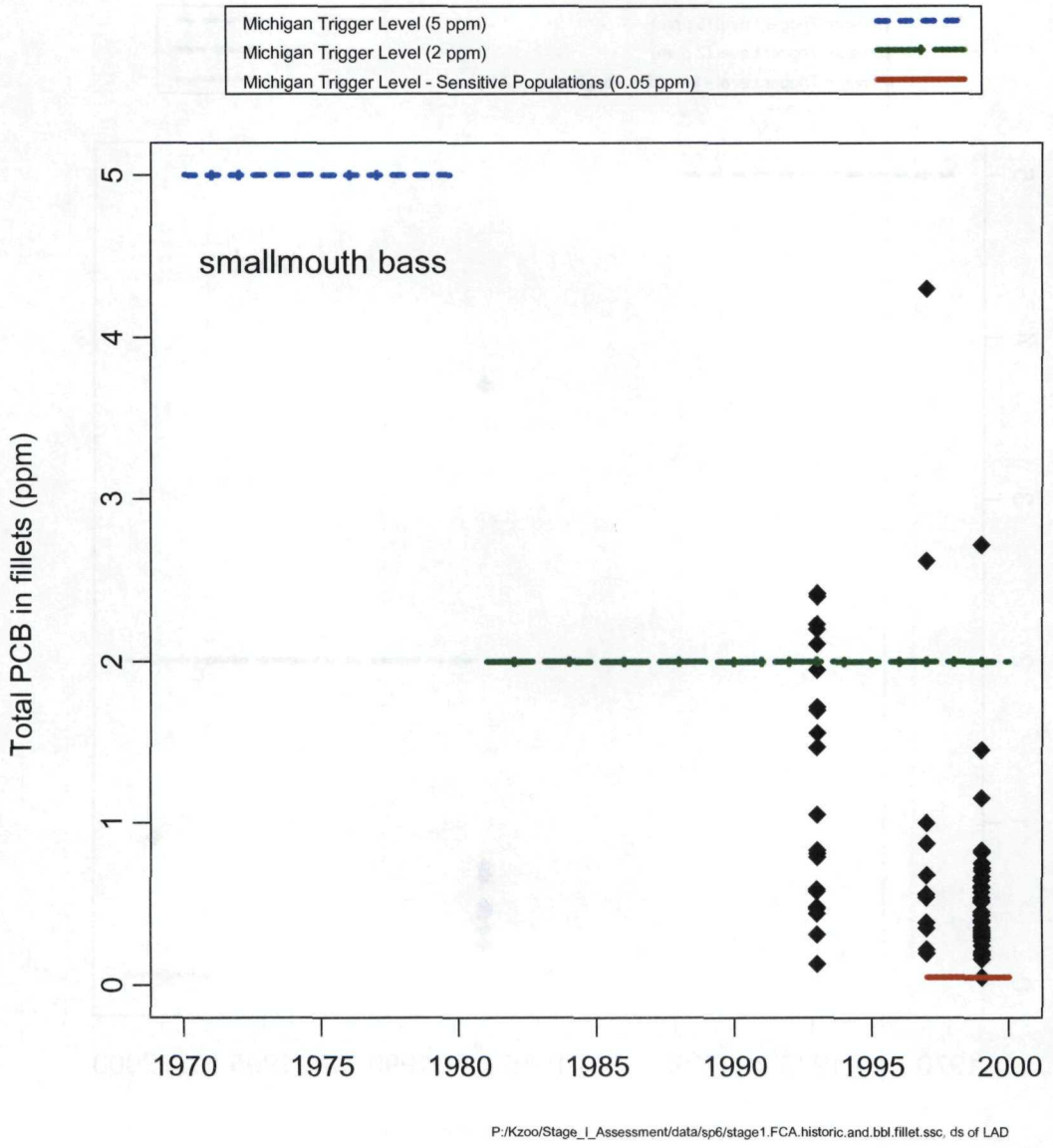

Figure 5.15. Total PCBs in fillets of smallmouth bass collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

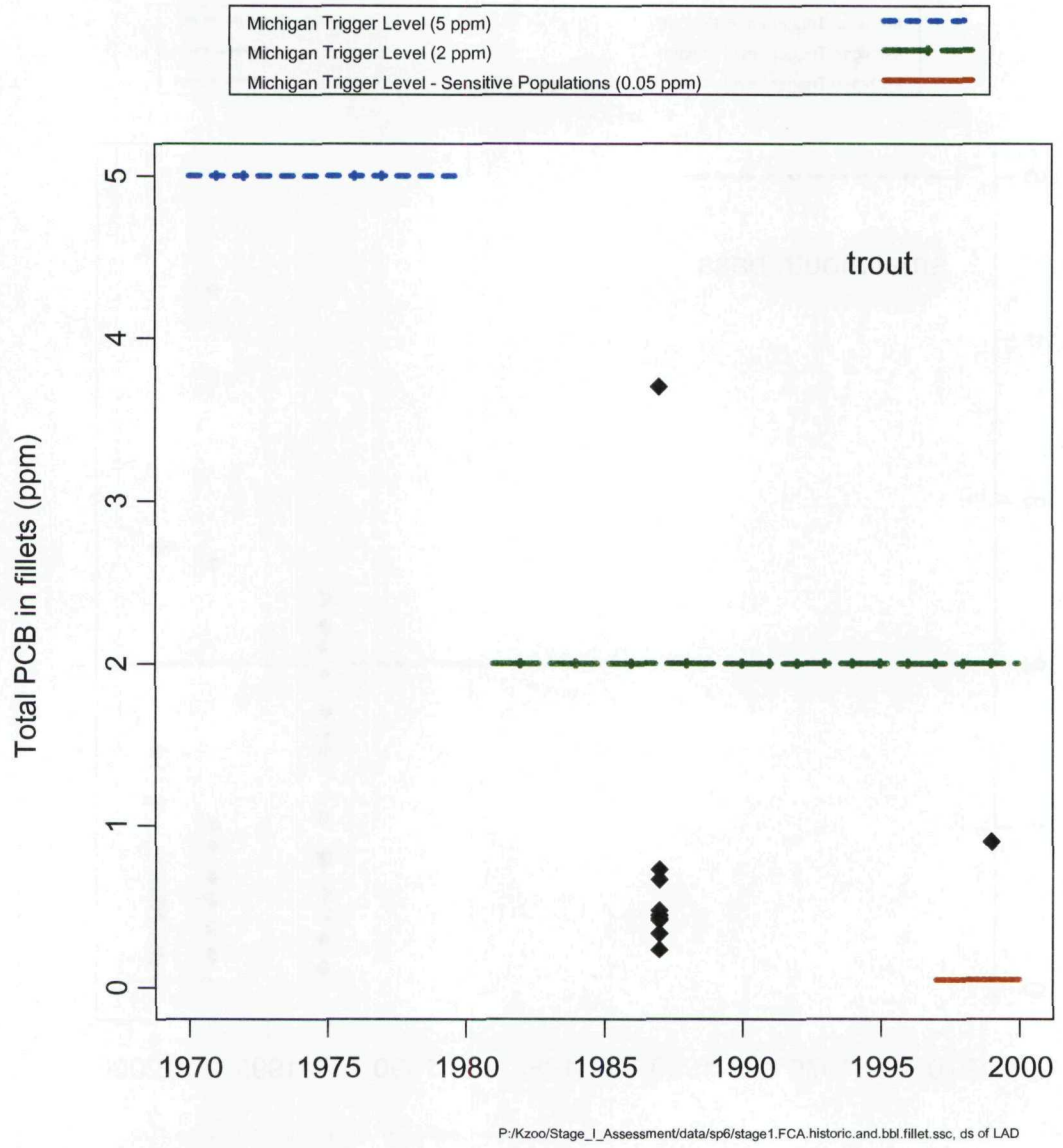

Figure 5.16. Total PCBs in fillets of trout collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

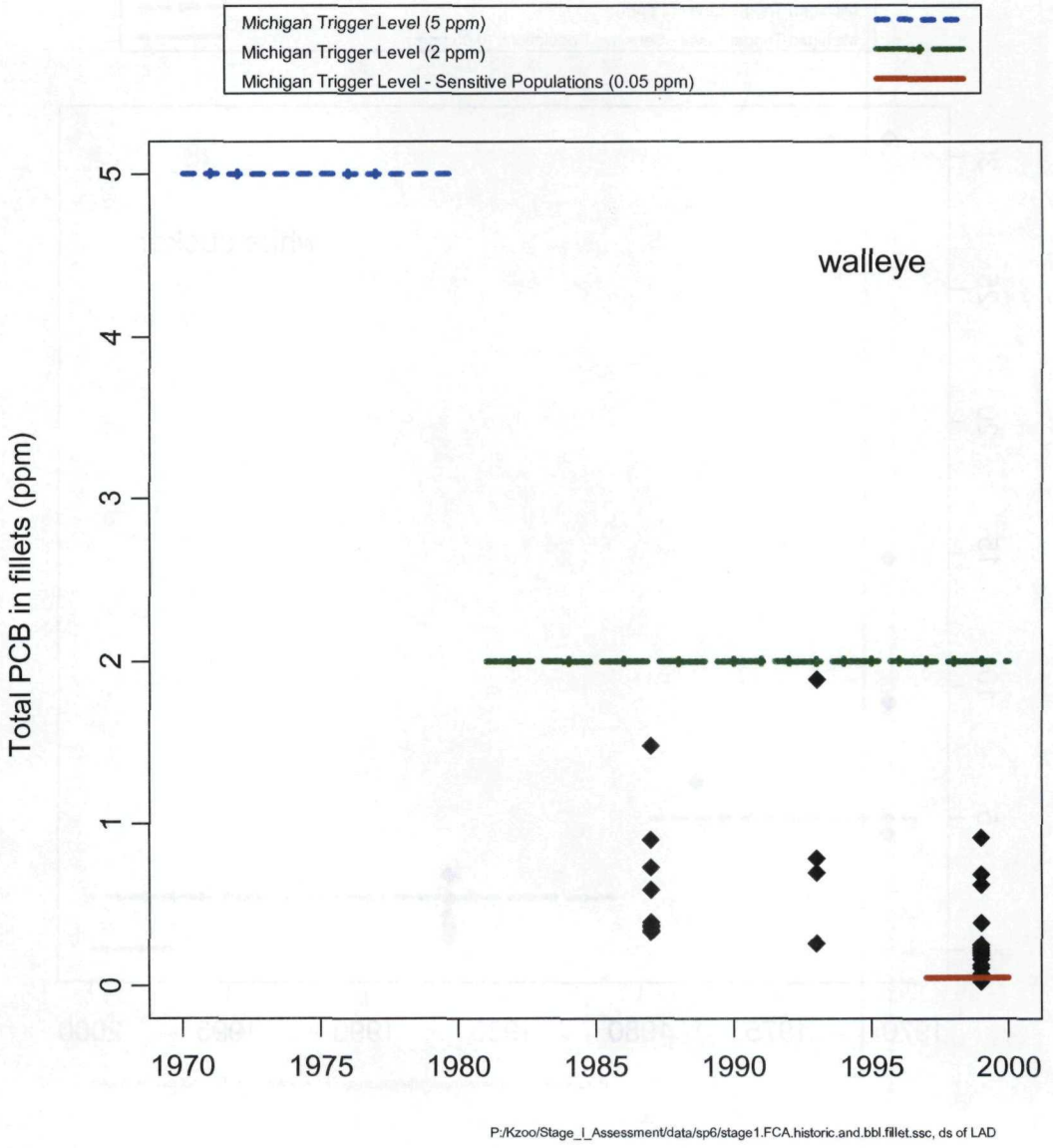

Figure 5.17. Total PCBs in fillets of walleye collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

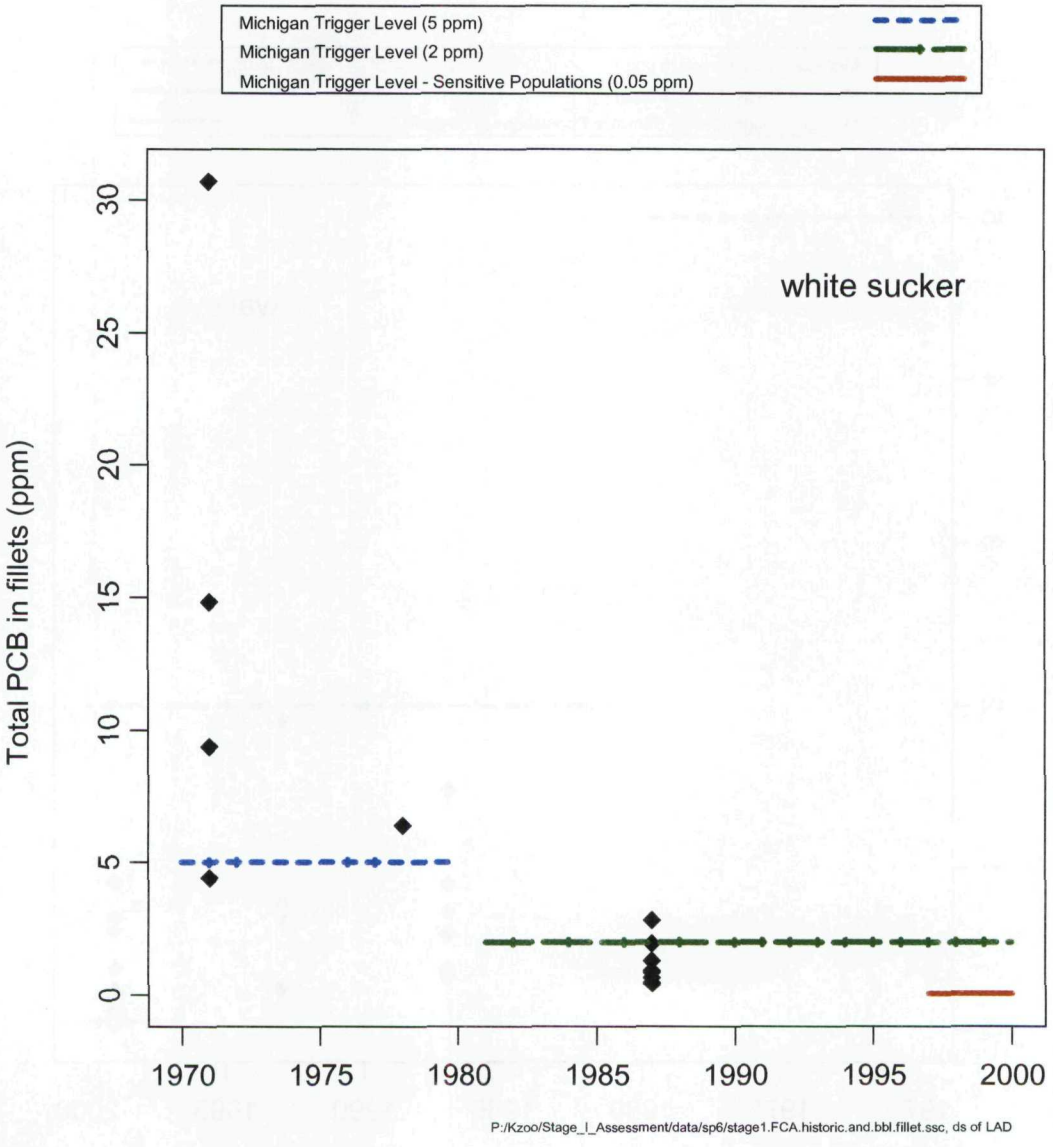

Figure 5.18. Total PCBs in fillets of white sucker collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

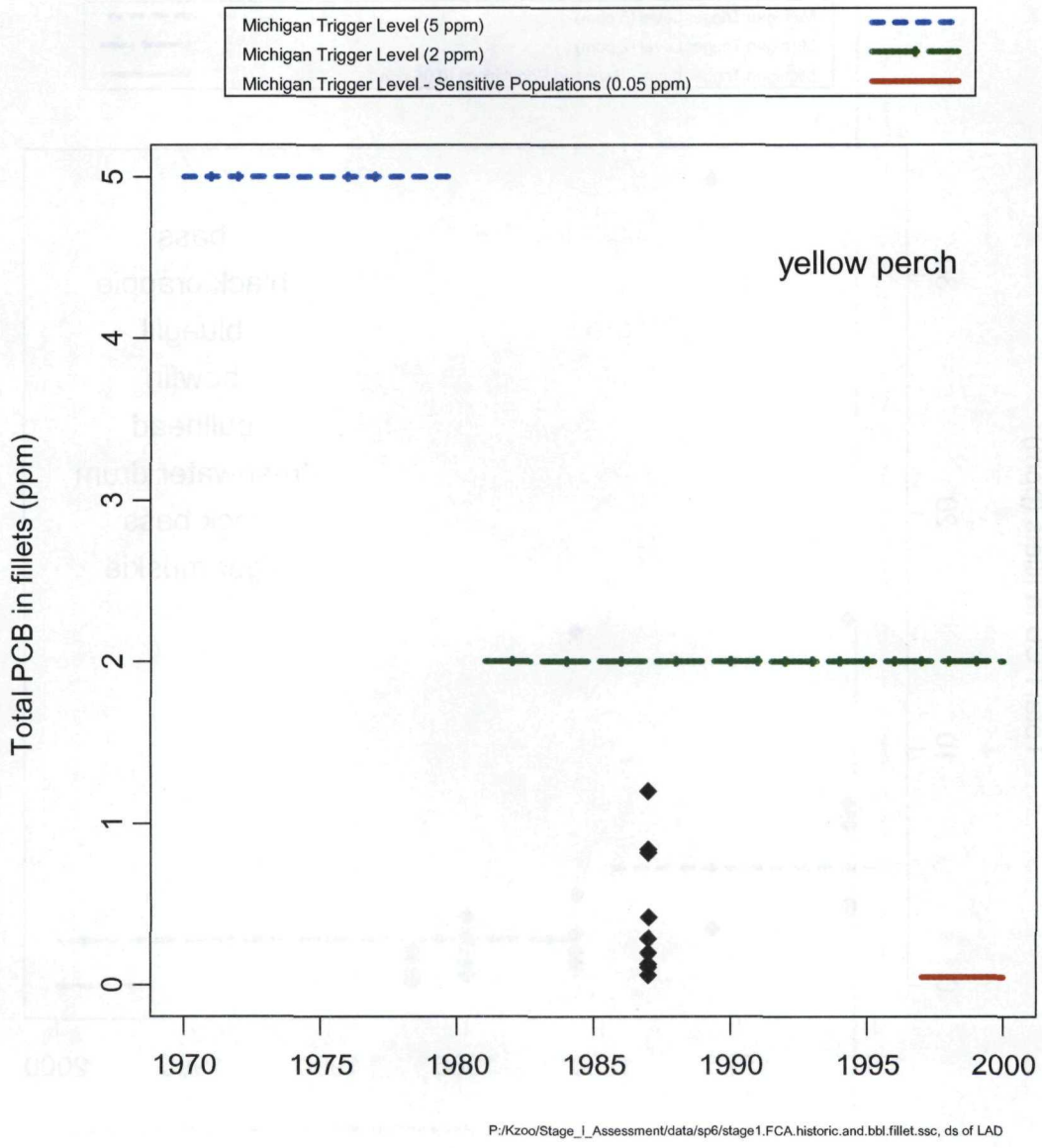

Figure 5.19. Total PCBs in fillets of yellow perch collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

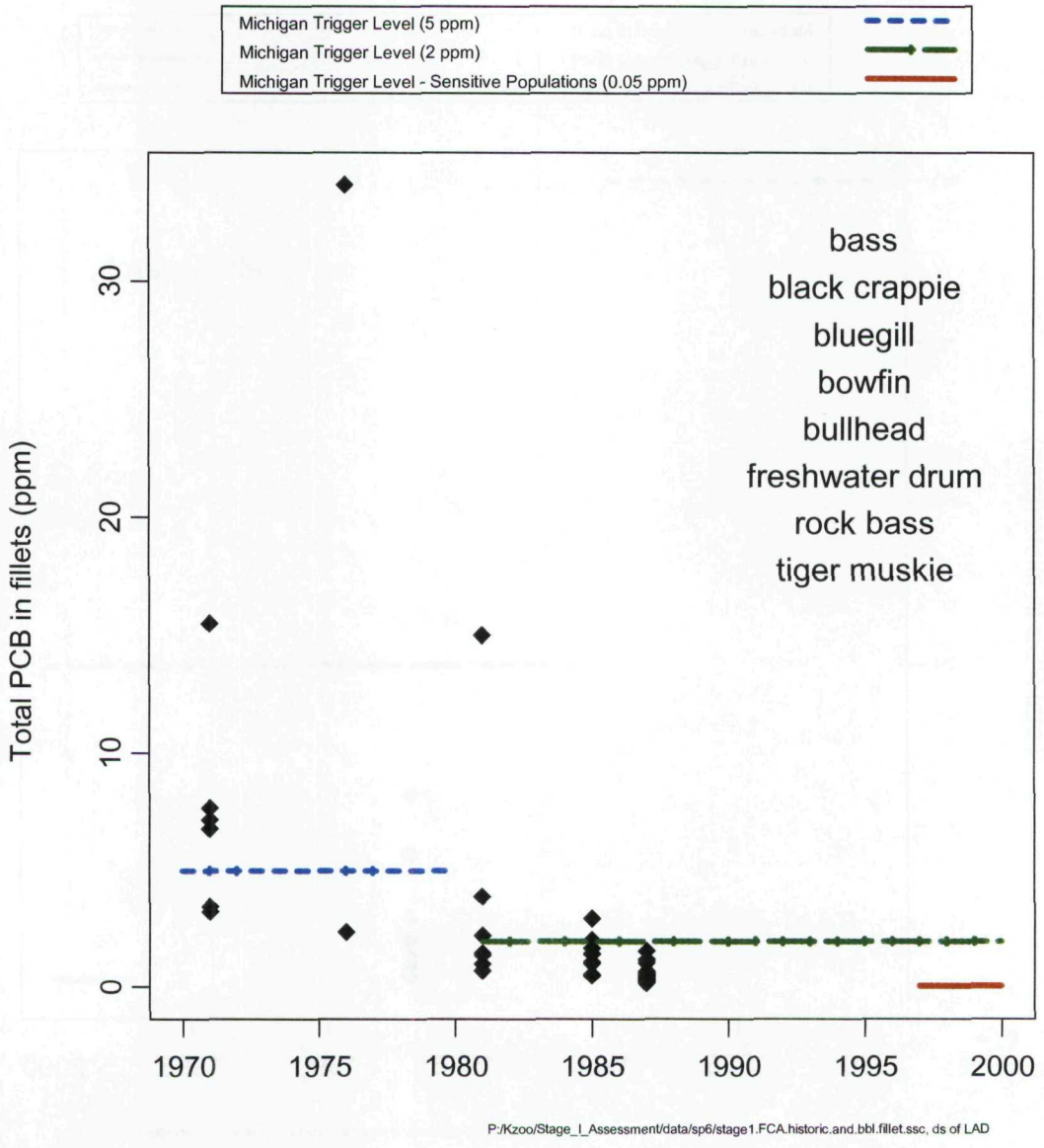

Figure 5.20. Total PCBs in fillets of other species under a general "all other species" advisory collected downstream of Lake Allegan Dam, 1971-2000.

Source: See Section 5.4.1.

walleye (Figure 5.17), and white sucker (Figure 5.18) exceeded trigger levels in place at the time of collection. In all of the samples from these species, only three walleye samples and one smallmouth bass sample collected in 1999 had PCB concentrations lower than the 0.05 ppm trigger level for sensitive populations. None of the yellow perch samples from 1987 exceeded the 2 ppm trigger level (Figure 5.19). Although the majority of samples from species that are under a general advisory in this reach (unknown species of bass, black crappie, bluegill, bowfin, bullhead, freshwater drum, rock bass, and tiger muskie) were below trigger levels, some samples did exceed them (Figure 5.20).

Overall, the tissue data indicate that Michigan's FCAs for the Kalamazoo River have a reasonable basis in the data. In all cases where data are available for species under a specific advisory in a given reach (see Tables 5.2 and 5.3), the total PCB concentration in fillets has exceeded trigger levels in some samples (Figures 5.3, 5.5-5.9, 5.11-5.18). Additionally, fillet samples from many species that fall under an "all other species" advisory exceeded the trigger levels at times (Figures 5.10 and 5.20). Samples collected from species that do not fall under any advisory were also found to exceed trigger levels (Figure 5.4). Ongoing monitoring conducted by MDEQ in 2001 and 2002 indicates that concentrations in fillets continue to exceed trigger levels (MDEQ, 2002, 2003).

5.5 Exceedences of the FDA Tolerance Level for PCBs

Fishery resources are injured if they contain concentrations of a hazardous substance sufficient to exceed action levels or tolerances established by the Food and Drug Administration (FDA) pursuant to the Food, Drug, and Cosmetic Act [43 C.F.R. § 11.62 (f)(1)(ii)]. This section compares measured PCB fillet concentrations with FDA tolerance levels.

5.5.1 Data sources

The data used to evaluate exceedences of the FDA tolerance level are the same used to evaluate exceedences of the Michigan FCA trigger levels and are described in Section 5.4.1.

5.5.2 Regulatory criteria and standards

FDA Tolerance levels are regulatory standards that are based on human health risk, while action levels are less formal administrative instructions that are more readily instituted and changed, and are generally replaced by a tolerance level (Boyer et al., 1991). The FDA proposed setting tolerance levels for PCBs in 1972 (Boyer et al., 1991), and established a temporary tolerance level of 5 ppm for PCBs in fish and shellfish in 1973 (38 Fed. Reg. 18096). At that time, the FDA stated that "the possibility of potential long-term hazards necessitates reduction of PCBs in

food as soon as possible." The advisories were considered "temporary" because "new data may justify a further downward revision of the tolerances" (42 Fed. Reg. 17493). The tolerance level was reduced to 2 ppm in 1984, based on further analysis of human health risks at different exposure levels (49 Fed. Reg. 21514). This reduction is codified at 21 C.F.R. § 109.30 (a)(7).

Therefore, PCB concentrations measured in edible portions of fish collected between 1973 and 1983 in the Kalamazoo River and Portage Creek are compared with the applicable FDA tolerance level of 5 ppm and concentrations in fish collected since 1984 are compared to the FDA tolerance level of 2 ppm.

5.5.3 Results

In most species collected between Battle Creek and Morrow Dam (upstream of PRP facilities), no or very few samples exceeded the FDA tolerance levels (Table 5.7). Carp fillets had PCB concentrations higher than these levels approximately 18% of the time. However, PCB concentrations in carp fillets have not exceeded the tolerance level since 1987. No samples from other species collected in this reach exceeded the tolerance level.

Samples of some species collected in the Kalamazoo River between Morrow Dam and Lake Allegan Dam and in Portage Creek exceeded the tolerance levels (Table 5.8). On average, 61% of carp fillets exceeded tolerance levels, and exceedences have persisted over time. Although fewer samples of other species have been collected, concentrations of PCBs in those fillets also exceeded the tolerance levels. Twenty-two percent of the fillet samples collected from channel catfish, 33% of largemouth bass, 23% of northern pike, 12% of smallmouth bass, 5% of walleye, and 42% of white sucker had PCB concentrations above the tolerance levels. Bass of unknown species, black crappie, bluegill, bullhead, pumpkinseed, rock bass, and trout had no samples with PCB concentrations exceeding the FDA tolerance levels in this reach.

As in the previous reach, many species sampled in the Kalamazoo River downstream of Lake Allegan Dam also exceeded FDA tolerance levels (Table 5.9). Carp and catfish had the highest concentrations and frequency of exceedence (68% and 73%, respectively). Fewer samples were collected of other species in this reach. However, 24% of bass of unknown species, 8% of largemouth bass, 15% of northern pike, 11% of smallmouth bass, 8% of trout, and 18% of white sucker fillets exceeded the tolerance levels. Of the species sampled, no black crappie, bluegill, bowfin, freshwater drum, rock bass, tiger muskie, walleye, or yellow perch fillets exceeded the tolerance levels.

Table 5.7. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River between Battle Creek and Morrow Dam

Species	Parameter[a]	1976	1981	1985	1986	1987	1993	1997	1999	2000
Bass	Number of samples	1	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	0.1	—	—	—	—	—	—	—	—
	% exceeding tolerance level	0.0%	—	—	—	—	—	—	—	—
Carp	Number of samples	1	11	20	19	18	22	22	33	11
	Maximum PCB conc. (ppm)	2.1	8.1	8.9	12.7	5.8	1.9	0.7	1.0	0.4
	% exceeding tolerance level	0.0%	18.2%	75.0%	52.6%	5.6%	0.0%	0.0%	0.0%	0.0%
Largemouth bass	Number of samples	—	2	3	—	1	—	—	—	—
	Maximum PCB conc. (ppm)	—	0.2	2.0	—	0.02	—	—	—	—
	% exceeding tolerance level	—	0.0%	0.0%	—	0.0%	—	—	—	—
Northern pike	Number of samples	1	1	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	3.7	0.1	—	—	—	—	—	—	—
	% exceeding tolerance level	0.0%	0.0%	—	—	—	—	—	—	—
Smallmouth bass	Number of samples	—	25	4	—	11	22	22	33	11
	Maximum PCB conc. (ppm)	—	1.8	1.4	—	1.3	0.7	0.3	1.1	0.03
	% exceeding tolerance level	—	0.0%	0.0%	—	0.0%	0.0%	0.0%	0.0%	0.0%
Walleye	Number of samples	—	—	—	—	—	3	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	0.3	—	—	—
	% exceeding tolerance level	—	—	—	—	—	0.0%	—	—	—
White sucker	Number of samples	1	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	2.0	—	—	—	—	—	—	—	—
	% exceeding tolerance level	0.0%	—	—	—	—	—	—	—	—

a. Percent exceeding tolerance level is based on a level of 5 ppm PCB before 1984 and 2 ppm PCB from 1984 on.

Table 5.8. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River between Morrow Dam and Lake Allegan Dam, and in Portage Creek

Species	Parameter[a]	1976	1978	1981	1983	1985	1986	1987	1993	1997	1999	2000
Bass	Number of samples	4	—	—	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	2.4	—	—	—	—	—	—	—	—	—	—
	% exceeding tolerance level	0.0%	—	—	—	—	—	—	—	—	—	—
Black crappie	Number of samples	—	—	3	—	—	—	—	—	—	1	1
	Maximum PCB conc. (ppm)	—	—	3.0	—	—	—	—	—	—	1.2	0.4
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	0.0%	0.0%
Bluegill (sunfish)	Number of samples	—	—	9	1	—	—	—	—	—	30	—
	Maximum PCB conc. (ppm)	—	—	1.3	0.5	—	—	—	—	—	0.8	—
	% exceeding tolerance level	—	—	0.0%	0.0%	—	—	—	—	—	0.0%	—
Bullhead (inc. black bullhead and yellow bullhead)	Number of samples	—	—	6	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	—	—	0.7	—	—	—	—	—	—	—	—
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	—	—
Carp	Number of samples	6	1	56	25	69	143	29	88	22	110	33
	Maximum PCB conc. (ppm)	13.8	51.3	47.0	15.9	14.0	27.4	17.1	17.2	17.3	21.7	10.3
	% exceeding tolerance level	100%	100%	25.0%	28.0%	87.0%	70.6%	62.1%	70.5%	31.8%	60.9%	27.3%
Channel catfish	Number of samples	—	—	1	—	—	—	—	—	—	17	—
	Maximum PCB conc. (ppm)	—	—	1.6	—	—	—	—	—	—	5.4	—
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	23.5%	—
Largemouth bass	Number of samples	—	—	6	1	7	—	—	—	—	—	1
	Maximum PCB conc. (ppm)	—	—	2.1	0.4	6.5	—	—	—	—	—	0.3
	% exceeding tolerance level	—	—	0.0%	0.0%	71.4%	—	—	—	—	—	0.0%

Table 5.8. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River between Morrow Dam and Lake Allegan Dam, and in Portage Creek (cont.)

Species	Parameter[a]	1976	1978	1981	1983	1985	1986	1987	1993	1997	1999	2000
Northern pike	Number of samples	1	1	5	—	—	—	3	—	—	15	1
	Maximum PCB conc. (ppm)	2.1	4.1	1.0	—	—	—	3.1	—	—	4.6	0.4
	% exceeding tolerance level	0.0%	0.0%	0.0%	—	—	—	66.7%	—	—	26.7%	0.0%
Pumpkinseed	Number of samples	—	—	1	—	—	—	—	—	—	14	—
	Maximum PCB conc. (ppm)	—	—	2.2	—	—	—	—	—	—	0.6	—
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	0.0%	—
Rock bass	Number of samples	—	—	1	—	—	—	—	—	—	—	1
	Maximum PCB conc. (ppm)	—	—	1.5	—	—	—	—	—	—	—	0.03
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	—	0.0%
Smallmouth bass	Number of samples	—	—	10	—	6	—	10	77	22	121	32
	Maximum PCB conc. (ppm)	—	—	2.2	—	3.3	—	5.1	5.8	1.6	5.1	2.3
	% exceeding tolerance level	—	—	0.0%	—	50%	—	70%	24.7%	0.0%	2.5%	3.1%
Trout	Number of samples	—	—	—	—	—	—	—	—	—	—	1
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	—	—	—	0.05
	% exceeding tolerance level	—	—	—	—	—	—	—	—	—	—	0.0%
Walleye	Number of samples	—	—	—	—	—	—	—	6	—	13	2
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	3.7	—	1.5	1.8
	% exceeding tolerance level	—	—	—	—	—	—	—	16.7%	—	0.0%	0.0%
White sucker	Number of samples	3	1	8	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	7.4	6.3	7.6	—	—	—	—	—	—	—	—
	% exceeding tolerance level	33.3%	100%	37.5%	—	—	—	—	—	—	—	—

a. Percent exceeding tolerance level is based on a level of 5 ppm PCB before 1984 and 2 ppm PCB from 1984 on.

Table 5.9. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River downstream of Lake Allegan Dam

Species	Parameter[a]	1976	1978	1981	1983	1984	1985	1986	1987	1993	1997	1999
Bass	Number of samples	1	—	6	—	—	10	—	—	—	—	—
	Maximum PCB conc. (ppm)	34.1	—	15.0	—	—	3.0	—	—	—	—	—
	% exceeding tolerance level	100%	—	16.7%	—	—	20.0%	—	—	—	—	—
Black crappie	Number of samples	—	—	—	—	—	—	—	10	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	1.6	—	—	—
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	—	—	—
Bluegill (sunfish)	Number of samples	—	—	—	—	—	—	—	10	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	0.7	—	—	—
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	—	—	—
Bowfin	Number of samples	—	—	2	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	—	—	2.3	—	—	—	—	—	—	—	—
	% exceeding tolerance level	—	—	0.0%	—	—	—	—	—	—	—	—
Carp	Number of samples	1	2	18	11	11	20	24	9	22	12	43
	Maximum PCB conc. (ppm)	36.0	63.0	16.0	25.7	25.7	9.1	10.5	8.6	17.0	17.3	6.9
	% exceeding tolerance level	100%	100%	55.6%	45.5%	63.6	80.0%	75.0%	88.9%	90.9%	66.7%	51.2%
Channel catfish (and flat catfish)	Number of samples	—	—	—	—	—	—	—	9	—	—	6
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	13.0	—	—	4.8
	% exceeding tolerance level	—	—	—	—	—	—	—	100%	—	—	33.3%
Freshwater drum	Number of samples	—	—	—	—	—	—	—	1	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	1.2	—	—	—
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	—	—	—

Table 5.9. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River downstream of Lake Allegan Dam (cont.)

Species	Parameter[a]	1976	1978	1981	1983	1984	1985	1986	1987	1993	1997	1999
Largemouth bass	Number of samples	—	—	8	—	—	—	5	10	—	—	2
	Maximum PCB conc. (ppm)	—	—	15.0	—	—	—	1.1	2.0	—	—	1.1
	% exceeding tolerance level	—	—	12.5%	—	—	—	0.0%	10.0%	—	—	0.0%
Northern pike	Number of samples	1	3	4	—	—	—	—	11	—	—	8
	Maximum PCB conc. (ppm)	4.7	10.9	0.8	—	—	—	—	3.4	—	—	0.5
	% exceeding tolerance level	0.0%	33.3%	0.0%	—	—	—	—	27.3%	—	—	0.0%
Rock bass	Number of samples	—	—	—	—	—	—	—	10	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	0.5	—	—	—
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	—	—	—
Smallmouth bass	Number of samples	—	—	—	—	—	—	—	—	22	11	40
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	—	2.4	4.3	2.7
	% exceeding tolerance level	—	—	—	—	—	—	—	—	22.7%	18.2%	2.5%
Tiger muskie	Number of samples	1	—	—	—	—	—	—	—	—	—	—
	Maximum PCB conc. (ppm)	2.4	—	—	—	—	—	—	—	—	—	—
	% exceeding tolerance level	0.0%	—	—	—	—	—	—	—	—	—	—
Trout (brown and rainbow)	Number of samples	—	—	—	—	—	—	—	11	—	—	1
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	3.7	—	—	0.9
	% exceeding tolerance level	—	—	—	—	—	—	—	9.1%	—	—	0.0%
Walleye	Number of samples	—	—	—	—	—	—	—	10	4	—	19
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	1.5	1.9	—	0.9
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	0.0%	—	0.0%

Table 5.9. Summary of PCB tissue concentrations and frequency of exceedences of FDA tolerance levels for fish in the Kalamazoo River downstream of Lake Allegan Dam (cont.)

Species	Parameter[a]	1976	1978	1981	1983	1984	1985	1986	1987	1993	1997	1999
White sucker	Number of samples	—	1	—	—	—	—	—	10	—	—	—
	Maximum PCB conc. (ppm)	—	6.4	—	—	—	—	—	2.8	—	—	—
	% exceeding tolerance level	—	100%	—	—	—	—	—	10.0%	—	—	—
Yellow perch	Number of samples	—	—	—	—	—	—	—	10	—	—	—
	Maximum PCB conc. (ppm)	—	—	—	—	—	—	—	1.2	—	—	—
	% exceeding tolerance level	—	—	—	—	—	—	—	0.0%	—	—	—

a. Percent exceeding tolerance level is based on a level of 5 ppm PCB before 1984 and 2 ppm PCB from 1984 on.

Review of the maximum PCB concentrations and the percentage of fillet PCB concentrations that exceed the FDA tolerance level indicates that the FDA tolerance has been and continues to be exceeded in multiple species in the Kalamazoo River between Morrow Dam and Lake Michigan, and in Portage Creek. Exceedences in these reaches have occurred since the establishment of the tolerance level in 1973 and have persisted through at least 2000.

5.6 Conclusions

Fish consumption advisories due to PCB contamination have been issued for many species in Portage Creek and the Kalamazoo River from Morrow Dam to Lake Michigan. Based on the extent and magnitude of PCB contamination in surface water and sediments throughout the KRE, the Trustees expect that consumption advisories will continue into the future. Although there is a consumption advisory for carp upstream of PRP facilities, advisories downstream of PRP facilities are more severe and apply to more species. The Trustees conclude that fish in Portage Creek and the Kalamazoo River have been injured according to the definition in 43 C.F.R. § 11.62(f)(1)(iii).

PCB concentrations in fish tissue have also exceeded tolerance levels established by the FDA under the Food, Drug and Cosmetic Act. A high percentage of samples exceeded the FDA tolerance levels in Portage Creek and the Kalamazoo River from Morrow Dam to Lake Michigan. Concentrations have exceeded these tolerance levels since the early 1970s and exceedences continue through 2000, the most recent year for which data are available. Based on the extent and magnitude of PCB contamination in surface water and sediments throughout the KRE, the Trustees expect that exceedences will continue into the future. The Trustees conclude that fish in Portage Creek and the Kalamazoo River have been injured according to the definition in 43 C.F.R. § 11.62(f)(1)(ii).

6. Injuries to Fish and Aquatic Invertebrates

This chapter presents the Stage I Injury Assessment for the fish and aquatic invertebrate resources of the KRE. The Kalamazoo River fish community upstream of the Lake Allegan Dam includes primarily carp, white sucker, smallmouth bass, walleye, northern pike, channel catfish, and black crappie (J. Wesley, MDNR Fisheries Division, personal communication, 2004). When dissolved oxygen in the river was low prior to the 1980s, carp and white suckers dominated the fishery (Knight and Lauff, 1969; MWRC, 1972a; Towns, 1984). Downstream of the Lake Allegan Dam, the fish community includes carp, smallmouth bass, largemouth bass, northern pike, channel catfish, flathead catfish (*Pylodictis olivaris*), black crappie, yellow perch, white sucker, freshwater drum, and some white bass/hybrid striped bass (Knight and Lauff, 1969; MWRC, 1972a; Towns, 1984; J. Wesley, MDNR Fisheries Division, personal communication, 2004). Additional species migrate upstream from Lake Michigan. Chinook salmon, coho salmon, rainbow trout, brown trout, walleye, and lake sturgeon run the lower river to spawn, and Lake Allegan Dam prevents passage of these fish to upstream areas. Stocking of salmonid species in this reach began in the early 1970s. Currently, chinook salmon, rainbow trout, brown trout, and walleye are stocked in the lower river (J. Wesley, MDNR Fisheries Division, personal communication, 2004).

Benthic invertebrates are important components of the aquatic food chain that live in close contact with river sediment. Many such invertebrates are the larval stages of insects, and mussels and other bivalves are also important components. Benthic invertebrates also play an important role in nutrient and energy cycling within the aquatic food chain. Their close contact with PCB contaminated sediment can make them relatively highly exposed to PCBs in the KRE, which coupled with their importance in the aquatic food chain, makes benthic invertebrates relevant to the Stage I assessment.

Ecosystem services provided by fish include prey for carnivorous and omnivorous wildlife, and nutrient and energy cycling. Human use services include fishing for recreation and as a food source. Benthic invertebrates provide important ecological services as well, as prey for fish, birds, and mammals, in nutrient cycling, and in energy transfer.

6.1 Injury Definitions

Biological resources are defined in the DOI regulations as "those natural resources referred to in section 101(16) of CERCLA as fish and wildlife and other biota. Fish and wildlife include marine and freshwater aquatic and terrestrial species; game, nongame, and commercial species; and threatened, endangered, and State sensitive species. Other biota encompass shellfish, terrestrial and aquatic plants, and other living organisms not listed in this definition" [43 C.F.R. § 11.14(f)]. This chapter addresses injuries to aquatic biota, specifically fish and aquatic invertebrates. Injuries to terrestrial wildlife are addressed in Chapter 7 of this document.

In this chapter, injuries to aquatic biota are determined using the following injury definition. According to DOI regulations, "an injury to a biological resource has resulted from the . . . release of a hazardous substance if concentration of the substance is sufficient to cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations" [43 C.F.R. § 11.62(f)(1)(i)].

An injury to biological resources can be demonstrated, per the DOI regulations, "if the biological response under consideration can satisfy all of the following acceptance criteria" [43 C.F.R. § 11.62 (f)(2): (i-iv)]:

- The biological response is often the result of exposure to . . . [the] hazardous substances [43 C.F.R. § 11.62 (f)(2)(i)].
- Exposure to . . . [the] hazardous substances is known to cause this biological response in free-ranging organisms [43 C.F.R. § 11.62 (f)(2)(ii)].
- Exposure to . . . [the] hazardous substances is known to cause this biological response in controlled experiments [43 C.F.R. § 11.62 (f)(2)(iii)].
- The biological response measurement is practical to perform and produces scientifically valid results [43 C.F.R. § 11.62 (f)(2)(iv)].

Injuries to biological resources may include death (e.g., fish kills), cancer (e.g., neoplasm), disease (e.g., fin erosion), physical deformation (e.g., deformities or lesions), behavioral abnormalities (e.g., avoidance), and physiological malfunctions (e.g., reduced reproduction) [43 C.F.R. § 11.62 (f)(4)].

6.2 Stage I Injury Assessment Approach

Exposure of fish to PCBs is known to cause a wide range of adverse effects (Table 6.1). Adverse effects in fish range from death (fry mortality and reduced egg survival rates) to sublethal and reproductive effects. PCBs have been found to promote the growth of cancerous tumors, and to reduce the effectiveness of the immune system in disease resistance. Physical deformations such as skeletal deformities and organ hemorrhaging have also been found in fish exposed to PCBs, particularly in fry. Physiological impairments caused by exposure to PCBs include endocrine system malfunction, decreases in fertility and other reproductive impairment, and biochemical changes. Chronic exposure of invertebrates to PCBs is known to cause reductions in growth and survival (Dillon et al., 1990; Eisler and Belisle, 1996; Ingersoll et al., 2000), even though invertebrates lack the aryl hydrocarbon receptor through which the coplanar PCB congeners act (West et al., 1997).

Table 6.1. Overview of adverse effects in fish caused by exposure to PCBs

Category	Response measure	Documented response	Example studies
Death	Mortality	Fry mortality and reduced egg hatchability	Nebeker et al., 1974; Eisler, 1986; Walker et al., 1994; Mac, 1988; Mac and Schwartz, 1992; Mac et al., 1993; Monosson et al., 1994
Cancer	Tumorigenesis	Tumor formation, especially in the liver	Hendricks et al., 1981, 1990; Baumann, 1992a, 1992b; Teh et al., 1997; Barron et al., 2000
		Enhanced tumorigenesis, via potentiation of estrogen responsiveness	Teh and Hinton, 1998
Disease	Immune system impairment	Reduced antibody levels	Thuvander and Carlstein, 1991
		Reduced immune cell activity	Jones et al., 1979; Arkoosh et al., 1994; Rice and Schlenk, 1995; Rice et al., 1996
		Reduced resistance to introduced bacteria	Jones et al., 1979
		Increased susceptibility to disease, parasitism, and cancer	Kahn and Thulin, 1991; Zelikoff, 1994; Anderson and Zeeman, 1995

Table 6.1. Overview of adverse effects in fish caused by exposure to PCBs (cont.)

Category	Response measure	Documented response	Example studies
Physical deformation	Deformities, especially in developing fry	Yolk sac edema	Walker et al., 1991
		Hemorrhaging in various organs	Spitsbergen et al., 1991; Walker and Peterson, 1992
		Skeletal deformation including domed skulls and craniofacial deformities	Walker et al., 1994
		Overt external malformations such as opercular defects	Helder, 1980, 1981
Physiological malfunction	Endocrine system and reproductive impairment	Promotion, inhibition, and mimicking of estrogens; competition with or alteration of thyroid hormone levels in the blood	Hansen, 1994; Gillesby and Zacharewski, 1998
		Altered sex determination and sex ratios	Matta et al., 1998
		Delayed maturity	Munkittrick et al., 1997
		Decreased fertility and egg production	Arcand-Hoy and Benson, 1998
		Gonadal abnormalities	Matta et al., 1998
		Reduced gonadal growth	Jobling et al., 1996
Physiological malfunction	Biochemical changes	Induced CYP1A activity, which is linked to tumorigenesis via metabolic activation	Van Der Oost et al., 1991; Wirgin et al., 1992; Stegeman and Hahn, 1994; Sleiderink et al., 1995; Eggens et al., 1996; Schrank et al., 1997; Courtenay et al., 1999

The approaches used in this chapter to assess injury to aquatic biota include comparisons of measured tissue PCB concentrations to toxicological benchmarks and in situ fish health studies (Table 6.2). The PCB concentrations measured in the last two decades in Kalamazoo River fish tissue are well below concentrations shown to cause direct mortality to adult fish (> 100 mg/kg ww) or adverse impacts to fish growth (> 50 mg/kg ww) (Niimi, 1996). Therefore, these endpoints are not assessed further. Instead, the Stage I assessment of injuries to fish focuses on embryomortality caused by PCBs deposited in fish eggs, and on histopathological or biochemical changes associated with PCB accumulation in fish livers. Fish are relatively sensitive to these

Table 6.2. Approaches to evaluate injury to aquatic biota

Injury definition	Stage I injury assessment approach	Chapter section
Cause the biological resource or its offspring to have undergone adverse changes in viability [43 C.F.R. § 11.62(f)(1)(i)].	Compare measured concentrations of PCBs in smallmouth bass and walleye eggs to toxicological benchmarks.	6.3
	Compare measured PCB concentrations in smallmouth bass livers to concentrations associated with adverse effects.	6.4
	Compare PCB concentrations and histopathological, immunological, and endocrine function parameters between fish in assessment locations and fish from reference locations.	6.5
	Compare surface sediment concentrations to consensus-based sediment effect concentrations for benthic invertebrates.	6.6

endpoints, and PCBs have been shown to cause these effects in laboratory studies and in field studies (Niimi, 1996). Embryomortality is assessed using a comparison of measured fish egg PCB concentrations with toxicity reference values from the literature. Histopathological and biochemical effects are assessed using a comparison of measured fish liver PCB concentrations with toxicity reference values from the literature and an evaluation of data collected on immunological, histopathological, and endocrine system function in KRE smallmouth bass. The data for these three evaluations come from the same study, a 1995 fish health study conducted by Stratus Consulting staff (Anderson et al., 2003). The Trustees are not aware of any other relevant studies of Kalamazoo River fish health or viability, nor of any other data on PCB concentrations in Kalamazoo River fish eggs or livers.

However, it should be noted that the data collected in the 1995 study are limited in species addressed, spatial coverage, sample size, and time period. A much larger amount of data is available for PCB concentrations (measured as Aroclors) in KRE fish fillets or whole bodies. However, using these data to assess potential injuries to fish necessarily involves uncertainties. No clear and generalizable relationship between PCB concentrations in fish fillet or whole bodies and sublethal adverse effects has been demonstrated across fish species and PCB composition (Monosson, 1999; Barron et al., 2002). Furthermore, the embryotoxicity of PCBs in fish eggs is believed to be caused by specific coplanar PCB congeners (see Section 6.3), and data from the 1995 study are the only data available on the concentrations of these congeners in KRE fish tissue. Furthermore, available data are not sufficient to reliably estimate across all of the sampled fish species and sample locations the concentrations of the coplanar PCB congeners in fish tissue from the available fish fillet and whole body Aroclor PCB data. Therefore, the Stage I Assessment of adverse effects injuries to fish is based on the site-specific data on PCB concentrations in fish eggs and livers from the 1995 study, and the larger dataset of PCB concentrations in fish fillets and whole bodies is not used.

This chapter also evaluates adverse effects to benthic invertebrates caused by releases of PCBs into the KRE (Table 6.2).

6.3 TCDD-Equivalents in Fish Eggs

In this section, measured concentrations of PCB congeners in fish eggs from the Kalamazoo River are compared to toxic thresholds for egg and fry mortality as a means of determining reproductive injuries to KRE fish. Fish eggs are the focus because developing embryos and fry are the most sensitive life stages to PCB toxicity, and because toxicity thresholds for PCBs in eggs are available from the literature (Cook et al., 1993; Peterson et al., 1993; Walker and Peterson, 1994; Eisler and Belisle, 1996). Many of the toxic effects of PCBs on developing fish are produced by specific coplanar PCB congeners that have a structure similar to that of 2,3,7,8-tetrachloro-*p*-dibenzodioxin (TCDD) and cause toxicity through a similar mechanism, and fish embryos and fry tend to be more sensitive to the TCDD-like toxicity of coplanar PCB congeners than they are to toxicity caused by other PCB congeners (Giesy and Kannan, 1998). TCDD has been studied in laboratory toxicity tests as the model compound for causing embryotoxicity to fish since it is the most potent compound at causing these effects. Therefore, to assess the reproductive toxicity of PCBs in fish eggs the Trustees use the dioxin equivalents approach, in which the concentrations of coplanar PCB congeners in fish eggs are converted to an equivalent concentration of TCDD. The dioxin equivalents approach to assessing the developmental toxicity of PCBs to fish is widely used and accepted (Van den Berg et al., 1998).

6.3.1 Data sources

The following data source was used in this evaluation:

- Study of fish health conducted by Stratus Consulting in 1995 (Anderson et al., 2003; analytical data in: A.D. Little, 1996; Midwest Research Institute, 1996).

As part of a larger study on smallmouth bass and walleye in the Kalamazoo River, Stratus Consulting collected eggs from 14 smallmouth bass and 7 walleye in the spring of 1995. Smallmouth bass samples were collected from below Morrow Dam, Verburg Park, Trowbridge Dam, and Lake Allegan. Walleye samples were collected at Lake Allegan. Eggs were taken from ripe females that were collected for tissue sampling. These egg samples were analyzed for a suite of 45 PCB congeners, including the coplanar PCB congeners PCB 77, 81, 126, and 169 (A.D. Little, 1996). However, the coplanar congener concentrations were below the detection limits of the analytical method used. Split samples of seven of the smallmouth bass and three of the walleye egg samples were analyzed separately for coplanar PCB congeners and polychlorinated dibenzodioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) using a high-resolution, low-detection analytical technique designed specifically for these congeners (Midwest Research Institute, 1996). Since the high-resolution method has lower detection limits and fewer potential interferences than the method used by A.D. Little, only the Midwest Research Institute data were used for these congeners.

6.3.2 Concentrations in eggs causing toxicity

Poor correlations have been observed between reproductive effects in fish and total PCB concentrations, especially concentrations reported in eggs (Williams and Giesy, 1992). This may be because of the range in toxicity of different PCB congeners and the variable proportion of congeners in PCB mixtures. To account for the variations in toxicity of different congeners, a PCB congener mixture can be converted to a toxicologically equivalent concentration of TCDD using toxicity equivalency factors (TEFs). TEFs are specific to each congener, and they represent the toxicity of the congener relative to TCDD. TCDD is used to calculate TEFs because it is the most toxic of the planar halogenated aromatic compounds, a group which includes PCDDs, PCDFs, and the coplanar PCB congeners. The TEF of a congener is determined by dividing the concentration of TCDD causing a given adverse response (e.g., concentration at which 50% mortality occurs) by the concentration of a congener causing the same level of response:

$$\text{TEF} = \frac{\text{TCDD concentration}}{\text{PCB congener concentration}}.$$

An international group of toxicology experts (Van den Berg et al., 1998) developed TEFs for several PCB and PCDD, and PCDF congeners based on their relative toxicity to fish (Table 6.3). These values were derived from multiple studies and are not specific to any single fish species.[1] Using these TEFs, contaminant concentrations in fish eggs can be converted to TCDD-equivalents (TCDD-eq). TCDD-eq is the concentration of TCDD that would have the same potency as the congener mix in a sample.

A TCDD-eq for a sample is calculated by summing the product of each congener's measured concentration and TEF in the sample (Giesy et al., 1994):

$$\text{TCDD-eq} = \sum([\text{congener}]_i \times \text{TEF}_i).$$

Calculated TCDD-eq concentrations can then be compared to TCDD concentrations that cause toxicity. Table 6.4 summarizes the toxic effects concentrations of TCDD in eggs, including no observed effect level (NOEL) and lowest observed effect level (LOEL). The concentrations at which effects are seen vary dramatically by species. The Giesy et al. (2002) chronic study on rainbow trout eggs produced the lowest LOEL in any published study to date of 0.1 pg/g ww in eggs. Other LOEL concentrations range from 40 pg/g for lake trout embryomortality to 3,330 pg/g for white perch embryomortality.

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1. TEFs specific to embryomortality in rainbow trout are available from several studies (Walker and Peterson, 1991; Zabel et al., 1995). However, the appropriateness of the TEFs from the rainbow trout studies to smallmouth bass and walleye is difficult to assess. Therefore, the more general TEFs from Van den Berg et al. (1998) are used here.

Table 6.3. Toxic equivalency factors of PCB congeners, PCDDs, and PCDFs relative to TCDD in fish eggs

Congener	TEF
PCB congener 77	0.0001
PCB congener 81	0.0005
PCB congener 126	0.005
PCB congener 169	0.00005
2,3,7,8 TCDD	1
2,3,7,8 TCDF	0.05
1,2,3,7,8 PeCDD	1
1,2,3,7,8 PeCDF	0.05
2,3,4,7,8 PeCDF	0.5
1,2,3,4,7,8 HxCDD	0.5
1,2,3,6,7,8 HxCDD	0.01
1,2,3,7,8,9 HxCDD	0.01
1,2,3,4,7,8 HxCDF	0.1
1,2,3,6,7,8 HxCDF	0.1
1,2,3,7,8,9 HxCDF	0.1
2,3,4,6,7,8 HxCDF	0.1
1,2,3,4,6,7,8 HpCDD	0.001
1,2,3,4,6,7,8 HpCDF	0.01
1,2,3,4,7,8,9 HpCDF	0.01
OCDD	0
OCDF	0

Source: Van den Berg et al., 1998.

Toxicity data can also be expressed as an LC50, which is the concentration at which a 50% mortality rate occurs. LC50s for lake trout eggs range from 58 to 80 pg/g TCDD, depending on exposure route (Walker et al., 1994). An LC50 concentration for brook trout eggs has been reported at 200 pg/g (Walker and Peterson, 1994) and LC50s for rainbow trout eggs range from 230 to 488 pg/g (Walker and Peterson, 1991). It should be noted that no LC50 value was derived in the Giesy et al. (2002) study because no consistent dose-response relationship existed. Eggs of other species that have been tested experience 50% mortality at TCDD concentrations of 250 pg/g or greater (Monosson et al., 1994; Elonen et al., 1998; Toomey et al., 2001).

Because of the range of the values shown in Table 6.4 and uncertainties with regard to species sensitivity, a single toxicity value is not derived from the values shown in the table. Rather, the calculated TCDD-eq concentrations in KRE fish eggs are compared to the entire range of reported effects concentrations.

Table 6.4. Effect endpoints based on TCDD concentrations in fish eggs (pg/g ww)

Species	Exposure route	NOEL[a]	LOEL	$LC_{egg}50$[b]	Source
Rainbow trout	Maternal, chronic exposure	—	0.1	—	Giesy et al., 2002
Lake trout	Maternal	23	50	58 (36-90)	Walker et al., 1994
Lake trout	Waterborne	34	40	69 (64-75)	Walker et al., 1994
Lake trout	Injection	44	55	80 (68-91)	Walker et al., 1994
Brook trout	Maternal	—	—	127 (106-145)	Johnson et al., 1998
Brook trout	Waterborne	135	185	200 (179-215)	Walker and Peterson, 1994
Rainbow trout, McConaughy strain	Injection	—	—	230 (208-249)	Walker and Peterson, 1991
Rainbow trout, Erwin strain	Injection	—	—	240 (209-264)	Walker and Peterson, 1991
Mummichog	Injection	—	—	250	Toomey et al., 2001
Rainbow trout, Arlee strain	Injection	—	—	374 (280-412)	Walker and Peterson, 1991
Rainbow trout, Eagle Lake strain	Injection	—	—	488 (338-580)	Walker and Peterson, 1991
Fathead minnow	Waterborne	235	435	539 (476-611)	Elonen et al., 1998
Channel catfish	Waterborne	385	855[c]	644 (576-721)	Elonen et al., 1998
Lake herring	Waterborne	175	270	902 (783-1,040)	Elonen et al., 1998
Medaka	Waterborne	455	949	1,110 (932-1,320)	Elonen et al., 1998
White sucker	Waterborne	848	1,220	1,890 (1,760-2,030)	Elonen et al., 1998
Northern pike	Waterborne	1,190	1,800	2,460 (2,100-2,880)	Elonen et al., 1998
Zebrafish	Waterborne	424	2,000	2,610 (2,310-2,950)	Elonen et al., 1998
White perch[d]	Injection	—	3,330	—	Monosson et al., 1994

a. For white sucker, effect endpoint was decreased growth, for all other species effect endpoint was decreased survival.
b. Concentrations in eggs causing 50% mortality to fish at test termination. Values in parentheses represent the 95% confidence interval, where available.
c. LOEL represents lowest experimental concentration in eggs where a significant decrease in mortality was observed. The next lowest exposure group had a concentration in eggs of 385 pg/g. The $LC_{egg}50$ of 644 pg/g was derived from a best fit concentration-response curve, and falls between these two values.
d. Concentration based on exposure to PCB-77 and converted to TCDD-eq using a TEF of 0.0001.

6.3.3 Results

The concentrations of TCDD-eqs from PCB, PCDD, and PCDF congeners in smallmouth bass and walleye egg samples are presented in Table 6.5. The highest concentrations are seen in smallmouth bass eggs collected from Verburg Park in the city of Kalamazoo and from Lake Allegan. Walleye eggs were collected only from Lake Allegan, and have lower concentrations of TCDD-eqs than smallmouth bass eggs from the same location.

Table 6.5. TCDD-equivalents of PCBs and PCDDs/PCDFs in fish eggs

Fish ID	Species	Location	TCDD-eq from PCBs (pg/g)[a]	TCDD-eq from PCDDs/PCDFs (pg/g)[b]	Total TCDD-eq (pg/g)
SMMP01	Smallmouth bass	Downstream of Morrow Lake	11.24	15.02	26.26
SMMP02	Smallmouth bass	Downstream of Morrow Lake	5.66	NA	NA
SMMP03	Smallmouth bass	Downstream of Morrow Lake	5.50	8.44	13.94
SMMP04	Smallmouth bass	Downstream of Morrow Lake	5.27	NA	NA
SMVP01	Smallmouth bass	Verburg Park	11.10	14.32	25.42
SMVP02	Smallmouth bass	Verburg Park	15.87	NA	NA
SMVP03	Smallmouth bass	Verburg Park	0.91	NA	NA
SMVP04	Smallmouth bass	Verburg Park	15.83	31.59	47.42
SMBT01	Smallmouth bass	Trowbridge Dam	3.65	NA	NA
SMLA01	Smallmouth bass	Lake Allegan	14.93	12.37	27.30
SMLD01	Smallmouth bass	Downstream of Lake Allegan	11.55	NA	NA
SMLD02	Smallmouth bass	Downstream of Lake Allegan	14.45	20.42	34.87
SMLD03	Smallmouth bass	Downstream of Lake Allegan	6.65	NA	NA
SMLD04	Smallmouth bass	Downstream of Lake Allegan	6.73	24.49	31.23
KZAD01	Walleye	Downstream of Lake Allegan	3.92	4.46	8.37
KZAD02	Walleye	Downstream of Lake Allegan	0.26	NA	NA
KZAD03	Walleye	Downstream of Lake Allegan	0.20	NA	NA
KZAD04	Walleye	Downstream of Lake Allegan	2.31	NA	NA
KZAD05	Walleye	Downstream of Lake Allegan	2.88	3.93	6.80
KZAD06	Walleye	Downstream of Lake Allegan	2.72	3.55	6.27
KZAD07	Walleye	Downstream of Lake Allegan	1.42	NA	NA

NA = sample not analyzed for these contaminants.

a. Calculated using the sum of TCDD-equivalents of detected PCB congeners.

b. Calculated using the sum of TCDD-equivalents of detected PCDDs and PCDFs.

Source: Midwest Research Institute, 1996.

When the concentrations in Table 6.5 are compared to the toxic effects endpoint concentrations in Table 6.4, TCDD-eq concentrations from PCBs are all lower than toxic effects thresholds, with the exception of the rainbow trout LOEL from the chronic exposure study conducted by Giesy et al. (2002). All of the fish egg samples contained TCDD-eq from PCBs at concentrations that exceed the 0.1 pg/g LOEL from this study. When the TCDD-eq concentration from PCBs is added to the TCDD-eq concentration from PCDDs and PCDFs, the total TCDD-eq concentrations are still less than the LOELs reported in Table 6.4, with the exception of the 0.1 pg/g LOEL from the Giesy et al. (2002) study.

It is difficult to draw conclusions regarding embryomortality injuries to fish from the available fish egg TCDD-eq data. Relevant data are available only for smallmouth bass and walleye eggs, and these data show that these two species are exposed to PCBs at concentrations lower than most of the available thresholds for embryomortality. However, the sensitivity of these two species (and most other KRE species) to embryomortality caused by PCBs or TCDD is not known. Fish species display widely variable sensitivity to TCDD toxicity. Lake trout are generally believed to be the most sensitive, although research by Cook (2000) suggests that bull trout may be more sensitive. In addition, the 0.1 pg/g long-term exposure LOEL for TCDD in rainbow trout eggs from the Giesy et al. (2002) study suggests that all of the sampled fish eggs contain PCBs at concentrations well above a LOEL. Finally, egg PCB data are available for KRE walleye and smallmouth bass only from 1995, and PCB exposures of these species (and others) in the past was higher than it was in 1995.

Therefore, the available data may indicate embryomortality injuries to KRE fish from PCB egg exposure. Additional information on the sensitivity of KRE species to PCB embryotoxicity and on the applicability of the Giesy et al. (2002) study results to KRE fish would help reduce the uncertainty in the injury determination.

6.4 Total PCBs in Smallmouth Bass Livers

In this section, PCB concentrations measured in livers of Kalamazoo River smallmouth bass are compared to concentrations associated with adverse sublethal effects from published literature, and to concentrations found to be associated with histopathological effects in walleye exposed to PCBs in Green Bay, Wisconsin. The amount of data available on PCB concentrations in KRE fish livers is small relative to the amount of whole body or fillet PCB concentration data. However, the liver is a site both of PCB accumulation and toxic action, and the relationship between adverse effects and PCB concentrations measured in livers is expected to be more robust than that for PCB concentrations in whole bodies or fillets (Monosson, 1999). Furthermore, although PCB concentrations measured in whole bodies or fillets could be converted to an estimated liver concentration on a lipid normalized basis (Russell et al., 1999), measurements of liver lipid content, which would be necessary to convert lipid normalized PCB concentrations in livers to equivalent wet weight concentrations for comparison to toxicological benchmarks, are not available for the fish sampled for whole body or fillet PCB concentrations. The liver lipid fractions measured in the Trustees' 1995 study are highly variable, making it unreliable to simply assume a single lipid fraction in all KRE fish livers. Therefore, the Trustees rely exclusively on the available liver PCB data for KRE fish from the Trustees' 1995 study.

6.4.1 Data sources

The following data source is used in this section:

- Study of fish health conducted by Stratus Consulting in 1995 (Anderson et al., 2003; analytical data in A.D. Little, 1996).

Twenty-nine smallmouth bass from assessment areas of the Kalamazoo River were collected and analyzed for PCBs (A.D. Little, 1996; State of Michigan Community Public Health Agency, 1996; Anderson et al., 2003). Livers from nine fish collected at D Avenue in Kalamazoo were collected and analyzed for 18 PCB congeners (8, 18, 28, 44, 52, 66, 101, 105, 118, 128, 138, 153, 170, 180, 187, 195, 206, 209; A.D. Little, 1996). Livers were also analyzed for coplanar PCB congeners (PCB 77, 81, 126, 169; Midwest Research Institute, 1996); however, there was insufficient tissue mass for obtaining detectable concentrations of these congeners at detection limits ranging from 0.0004 to 0.007 mg/kg ww (Anderson et al., 2003). The sum of the 18 PCB congeners was calculated using a value of zero where individual congeners were not detected.[2] An estimate of total PCB concentration in each liver sample was made from the sum of 18 measured congeners using a linear regression model.[3]

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2. Non-detects were infrequent and occurred at detection limits less than 0.005 mg/kg, except for congener number 209, decachlorobiphenyl. This congener was not detected in any sample at detection limits up to 0.12 mg/kg, but this congener occurs at non-measurable amounts in Aroclor mixtures (Frame et al., 1996). Therefore, the use of zero for non-detects introduces only negligible bias to the analysis.

3. The relationship between the sum of 18 measured congeners and total PCBs was developed from whole body fish tissue data from the Kalamazoo River, described in Section 7.4.1 of this report (Michigan State University Aquatic Toxicology Laboratory, 2002j). In that study seventy-six PCB congeners were measured in fish tissue samples, including the 18 congeners measured in smallmouth bass livers in the Trustees' study. The data from the Michigan State University study can thus be used to develop a relationship for Kalamazoo River fish between the sum of the 18 congeners and the sum of the 76 congeners. The relationship is characterized by the equation: $sum_{76\ congeners}(mg/kg) = 2.18 \times sum_{18\ congeners}(mg/kg)$ (multiple R-squared = 0.9923, residual standard error = 0.458 mg/kg). The Trustees assumed that these 76 congeners represented total PCBs, and used this equation (forced through an intercept of zero) to estimate total PCBs in the Kalamazoo River fish sampled by the Trustees. The Trustees evaluated the assumption that the sum of the 76 congeners can be used to represent total PCBs by reviewing PCB concentration data for 112 congeners in biota collected in the Lower Fox River and Green Bay, Wisconsin. Although the Wisconsin biota dataset contained some different coelutions of PCB congeners, 72 of the 76 congeners reported in the Michigan State University Kalamazoo River data also were measured in the Wisconsin study. On average, the sum of the 72 congeners was approximately 91% of the sum of the 112 congeners measured in the Wisconsin study. Additionally, Laurenstein et al. (1993) report that total PCB concentrations as the sum of homologues are approximately two times the sum of the 18 PCB congeners which were measured in the Kalamazoo River smallmouth bass livers.

6.4.2 Toxicological benchmarks

Table 6.6 lists available studies from the literature that exposed fish to PCBs in a laboratory setting and observed adverse sublethal affects that are associated with a PCB concentration accumulated in the liver. There are few toxicological benchmark values available for PCBs in fish livers. Although numerous toxicological studies are available from which fish liver PCB concentrations associated with toxicity could be developed, most of the studies are on individual PCB congeners, from field studies where other contaminants are also present, or rely on rough approximations to estimate liver PCB concentrations. Table 6.6 lists the results from available laboratory studies where fish were dosed with a PCB mixture and LOEL values for measured PCB concentrations in fish livers can be obtained. Other studies are available with TCDD (e.g., Walter et al., 2000), but KRE fish contaminant data are insufficient to estimate TCDD-eq in fish livers. The effects concentrations for PCBs in fish liver shown in Table 6.6 range from approximately 24.2 mg/kg for reduced fecundity, egg hatching rate, and alterations in liver microstructure in barbel to 45-100 mg/kg for alterations in hormone levels and testicular growth in Atlantic cod. Based on the studies listed in Table 6.6, the Trustees use the lowest reported LOEL concentration of 24.2 mg/kg PCBs in fish liver as a laboratory study-based injury threshold for fish. However, it should be noted that few directly relevant studies have been reported in the literature.

Table 6.6. PCB concentrations in livers of adult fish associated with adverse effects in laboratory studies[a]

Species	PCB mixture	Liver PCB concentration (mg/kg ww)	Adverse effect	Study
Barbel	Aroclor 1260	24.2[b]	Reduced fecundity; reducing egg hatching rate; liver ultrastructure alterations	Hugla and Thome, 1999
Atlantic croaker	Aroclor 1254	25	Decreased serotonin (by 38%), dopamine (by 35%), testicular growth (by 79%), testosterone (by 60%), 11-ketostosterone (by 73%); inhibition of gonadotropin secretion	Khan and Thomas, 1996, 1997
Atlantic cod	Aroclor 1254	45-100	Abnormal testes; altered steroid hormone metabolism; decreased spermatogenic elements	Sangalang et al., 1981; Freeman et al., 1982
Chinook salmon	Aroclor 1254	54	Immune system suppression	Arkoosh et al., 1994

a. Only studies where PCBs were directly measured in the liver are shown. Data from Black et al. (1998), in which killifish were injected with PCBs, are not included because the injection was done with a unique mixture of 8 PCB congeners and it is difficult to compare the results with the measurements of PCBs in KRE fish. A qualitative evaluation of the data indicates that the fish in the Black et al. (1998) study were exposed to higher levels of PCBs than measured in KRE bass.

b. Value is calculated from a mean dry weight concentration of 6.3 mg/kg dw from the paper using an assumed moisture content of 74% (Connolly et al., 1992).

Most of the studies listed in Table 6.6 used Aroclor 1254 as the PCB dosing mixture. Although the specific mixture of PCB congeners in the KRE is not the same as in Aroclor 1254, the general pattern of the congeners present in fish tends to match the general pattern of Aroclor 1254 (Blasland, Bouck & Lee, 2000c). This occurs even though the bulk of the PCBs released from the paper company facilities was Aroclor 1242 because of changes in the congener pattern that can occur once the PCBs were released (Erickson, 1997; Cacela et al., 2002). Therefore, although the PCB mixture used in the laboratory does not match the mixture that was originally released to the KRE , the general congener pattern of the mixtures used in the laboratory is similar to that found in fish from the KRE.

Additional benchmarks are drawn from a field study on adverse effects in walleye exposed to PCBs in the Fox River and Green Bay, Wisconsin (Barron et al., 2000). The Fox River/Green Bay environment is similar to the KRE in that it has been contaminated by releases of Aroclor 1242 from paper companies involved in the production or deinking of carbonless copy paper containing PCBs. This study found a significantly greater prevalence of cancerous tumors and precancerous lesions in the livers of walleye from assessment areas than in the livers of walleye from reference areas in Lake Winnebago and Patten Lake ($p = 0.004$). After removal of sections for histopathological analysis and disease screening, the remaining liver samples were analyzed for PCB concentrations as Aroclors. Mean concentrations of total PCBs in the livers of Fox River and Green Bay fish ranged from 3.6 to 6.4 mg/kg ww, and were significantly greater than concentrations in reference areas ($p < 0.01$) (Table 6.7). This range of PCB concentrations in fish livers associated with adverse effects is similar to the results of Mikaelian et al. (2002), who observed liver tumors and pre-neoplastic lesions in lake whitefish from the St. Lawrence River with mean liver PCB concentrations of 1.75 mg/kg (quantified as Aroclor 1254).

6.4.3 Results

Estimated total PCB concentrations in the 9 smallmouth bass livers collected from Kalamazoo River fish range from 1.68 to 12.80 mg/kg ww with an arithmetic mean concentration of 4.77 mg/kg ww and a geometric mean concentration of 3.83 mg/kg ww (Table 6.8). These concentrations are less than liver Aroclor 1254 concentrations reported in laboratory studies as being associated with adverse sublethal effects (Table 6.6). However, few laboratory studies are available that have assessed the sublethal impacts of PCBs to fish and simultaneously measured liver PCB concentrations, and no studies are available for species of interest in the KRE. Thus, a comparison of measured PCB concentrations in KRE fish livers with laboratory toxicity data suggests that concentrations in KRE fish are less than effects levels, but the comparison is limited in scope.

Table 6.7. Mean and SD of concentrations of total PCBs in walleye livers collected in the Lower Fox River and Green Bay, Wisconsin, in 1996 and 1997, and associated prevalence of foci of cellular alteration and hepatic tumors

Sample location	Number of samples	Total PCB mg/kg ww mean (SD)	Total PCB mg/kg (lipid) mean (SD)	Prevalence of foci of cellular alteration	Prevalence of hepatic tumors
Reference areas					
Lake Winnebago	12	0.94 (0.55)	27.0 (18.0)	10%	0%
Patten Lake	13	0.02 (0.07)	0.47 (1.7)	0%	0%
Assessment areas: Fox River/Green Bay					
Lower Fox River	20[a]	6.4 (1.7)	61.8 (13.7)	8%	4%
Lower Green Bay	16	4.3 (3.4)	44.3 (37.6)	25%	6%
Eastern Green Bay	21	3.6 (2.7)	36.4 (17.9)	24%	5%
Western Green Bay	18	4.0 (2.0)	33.3 (10.2)	33%	11%
Upper Green Bay	4	4.4 (1.4)	32.4 (9.5)	0%	50%

a. Number of samples indicates the number of separate sample analyses. Analyses were of 19 individual fish livers and 1 composite sample of livers from four fish.

Source: Barron et al., 2000.

Table 6.8. Total PCB concentrations in smallmouth bass livers from D Avenue, Kalamazoo

Fish ID	Sum of 18 PCB congeners in liver (mg/kg ww)	Estimated total PCBs in liver (mg/kg ww)
SBDA01	1.90	4.15
SBDA02	1.03	2.25
SBDA05	5.87	12.80
SBDA06	3.14	6.85
SBDA09	0.80	1.68
SBDA10	1.87	4.07
SBDA11	0.93	2.02
SBDA12	3.16	6.88
SBDA15	1.04	2.26
Arithmetic mean	2.19	4.77
Geometric mean	1.76	3.83

Source: A.D. Little, 1996.

The mean estimated concentration of total PCBs in Kalamazoo River smallmouth bass livers, 4.70 mg/kg ww, is within the range of the lowest mean concentrations observed in the livers of Green Bay walleye with increased prevalence of foci of cellular alteration and hepatic tumors (3.6 mg/kg ww in livers of fish from eastern Green Bay to 6.4 mg/kg ww in livers of fish from the lower Fox River).

These data indicate that the PCB concentrations in KRE smallmouth bass livers are similar to those observed in Green Bay walleye (Figure 6.1). Therefore, depending on the PCB sensitivity of smallmouth bass compared to walleye, KRE smallmouth bass may be exposed to PCBs at concentrations associated with liver tumors and pre-tumors. In the next section, the results of an investigation of the health of KRE smallmouth bass are presented and discussed.

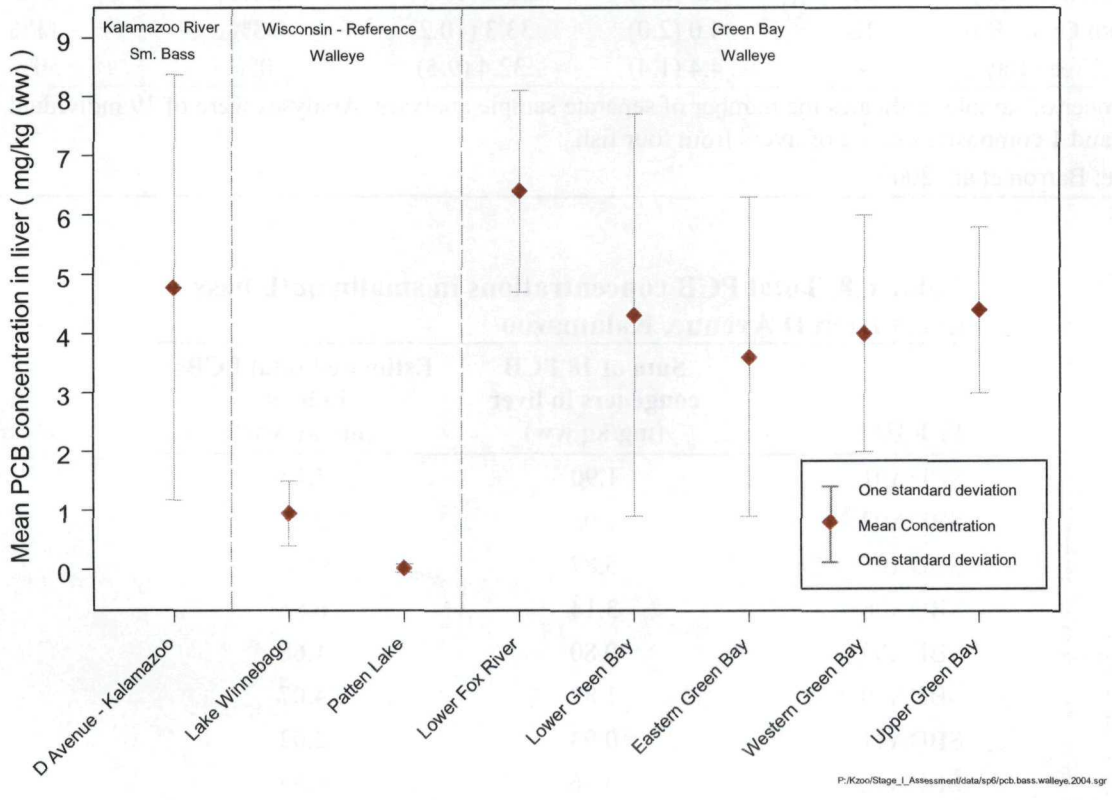

Figure 6.1. Mean and SD of concentrations of estimated total PCBs ww in smallmouth bass livers collected in the Kalamazoo River in 1995 and total PCBs in walleye livers collected in the Lower Fox River and Green Bay, Wisconsin, in 1996 and 1997.

Sources: A.D. Little, 1996; Barron et al., 2000.

6.5 Smallmouth Bass Condition, Biochemical, and Histopathological Status

6.5.1 Data sources

The following data source is described and discussed in this section:

- Study of smallmouth bass health conducted by the Trustees in 1995 (Anderson et al., 2003).

Thirty smallmouth bass, aged 3 to 5 years, were collected from the Kalamazoo River in 1995 using electroshocking (Anderson et al., 2003). Half of the bass were sampled from an upstream area near Ceresco, and half were sampled from an area near D Avenue in Kalamazoo. The upstream area served as a reference area because sediments of the upstream area were known to be relatively uncontaminated with PCBs (Blasland, Bouck & Lee, 2000b). The presence of Morrow Dam (Figure 1.1) prevents fish from migrating from the assessment area to the upstream area, but downstream migration is not prevented.

Bass were euthanized and samples were collected for a suite of analyses designed to evaluate health parameters that can be adversely affected by exposure to PCBs. Blood was collected for analysis of plasma vitellogenin, a protein produced by females that is used in the formation of eggs and is an indicator of estrogenic or antiestrogenic effects in fish. Liver tissue samples were analyzed for ethoxyresorufin-O-deethylase (EROD) activity, an enzyme that is involved in detoxifying pollutants, and for the enzyme cytochrome P4501A (CYP1A), a protein involved in the metabolism of planar aromatic hydrocarbons, including PCBs. Liver and spleen tissue samples were analyzed for superoxide dismutase activity (SOD), a critical antioxidant enzyme. Finally, various tissues (gill, liver, spleen, head kidney, trunk kidney, thyroid, and gonad) were histopathologically examined. Fillet samples were collected for analysis of PCB as Aroclors. Additionally, the livers of 9 bass from the assessment area and 10 bass from the reference area were analyzed for the concentrations of 18 PCB congeners. Total PCBs in livers was estimated from the sum of the 18 congeners using the method described in Section 6.4.1. PCB concentrations in both livers and fillets were normalized by lipid content and both wet weight and lipid normalized concentrations were used in further analyses.

6.5.2 Summary of results

Means of sum of PCB congeners in livers and total PCBs in fillets are significantly ($p < 0.001$) elevated in the assessment area compared to the reference area (Table 6.9). When PCB concentrations are lipid-normalized, liver sum of PCB congeners are positively correlated with fillet total PCBs across all fish ($p < 0.001$, rho = 0.85).[4]

Table 6.9. PCB concentrations in liver and fillet of smallmouth bass from the Kalamazoo River, 1995

	Liver					Fillet		
		Sum of 18 PCB congeners (mg/kg ww)[b]		Estimated total PCB concentration (mg/kg ww)[c]			Total PCB concentration in fillet (mg/kg ww)[d]	
Location	N[a]	Mean	SD	Mean	SD	N[a]	Mean	SD
Ceresco (reference)	10	0.26	0.10	0.60	0.21	15	0.09	0.04
D Avenue (assessment)	9	2.19[e]	1.65	4.77	3.60	15	1.01[e]	0.43

a. N = number of samples. Fewer liver samples were obtained for PCB analysis than for fillet analysis because liver mass was sometimes insufficient.
b. As reported in Anderson et al., 2003.
c. Total PCB concentration estimated from results of 18 congeners (see Section 6.4.1 for details).
d. Skin-on fillet total PCBs estimated by Aroclor method.
e. Significantly different than reference ($p < 0.001$).
Source: Anderson et al., 2003.

Bass in the assessment area have statistically significant differences in body condition and biochemical status compared to reference area bass (Table 6.10). Overall indices of fish health such as liver organosomatic index (liver weight as a percentage of total body weight) and condition factor (body weight divided by length cubed) are significantly lower in assessment area bass compared to reference area bass. In pooled samples from both sites, these indices were negatively correlated with liver and fillet PCB concentrations.

4. rho = Spearman's rank correlation.

Table 6.10. Summary of body condition and biochemical results for KRE smallmouth bass and correlations with tissue PCB concentrations

Parameter	Statistical significance[a,b]	Significant correlations with sum of PCB congeners on wet weight and lipid bases in liver		Significant correlations with total PCBs on wet weight and lipid bases in fillet	
		ww	Lipid	ww	Lipid
General indices					
Length	Not sig.	—[c]	—	—	—
Weight	Not sig.	—	—	—	—
Gender	Not sig.	—	—	—	—
Age	Not sig.	—	—	—	—
Spleen organosomatic index[d]	Not sig.	—	—	—	—
Testis organosomatic index[d]	Not sig.	—	—	—	—
Ovary organosomatic index[d]	Not sig.	—	—	—	—
Liver organosomatic index[d]	$a < r$ ($p < 0.05$)	Negative ($p = 0.05$, rho = -0.45)	Not sig.	Not sig.	Negative ($p < 0.01$, rho = -0.50)
Condition factor	$a < r$ ($p < 0.001$)	Negative ($p = 0.001$, rho = -0.77)	Negative ($p < 0.01$, rho = -0.64)	Negative ($p < 0.02$, rho = -0.46)	Negative ($p < 0.001$, rho = -0.70)
Biochemical indices					
Liver EROD (males)	$a < r$ ($p = 0.003$)	Negative ($p = 0.022$, rho = -0.76)	Negative ($p = 0.035$, rho = -0.70)	Negative ($p = 0.032$, rho = -0.57)	Negative ($p = 0.043$, rho = -0.54)
Liver EROD (females)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.
Liver CYP1A concentration (males)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.
Liver CYP1A concentration (females)	Not sig.	Not sig.	Not sig.	Not sig.	Not sig.

Table 6.10. Summary of body condition and biochemical results for KRE smallmouth bass and correlations with tissue PCB concentrations (cont.)

Parameter	Statistical significance[a,b]	Significant correlations with sum of PCB congeners on wet weight and lipid bases in liver		Significant correlations with total PCBs on wet weight and lipid bases in fillet	
		ww	Lipid	ww	Lipid
SOD activity in spleen	a < r (p = 0.001)	Negative (p = 0.016, rho = -0.56)	Negative (p = 0.029, rho = -0.51)	Not sig.	Negative (p = 0.03, rho = -0.40)
SOD activity in liver	a < r (p = 0.015)	Negative (p = 0.026, rho = -0.52)	Negative (p = 0.005, rho = -0.66)	Negative (p = 0.002, rho = -0.58)	Negative (p = 0.001, rho = -0.60)
Plasma vitellogenin (females)	a < r (p < 0.05)	Not sig.	Negative (p = 0.035, rho = -0.79)[e]	—	—

a. a = assessment, r = reference.
b. Not sig. = not significant, p > 0.05.
c. — = not reported.
d. Organosomatic index = (organ weight/body weight) × 100.
e. Correlation using only females with mature oocytes (three fish from reference area were eliminated from this analysis because they contained predominately immature oocytes).

Source: Anderson et al., 2003.

Biochemical indices previously shown to be associated with or modulated by PCBs include liver EROD activity, liver CYP1A relative protein concentrations, liver and spleen SOD activity, and plasma vitellogenin concentrations (Anderson et al., 2003). Liver EROD activity in male assessment area bass is significantly lower than in male reference area bass, but there is no significant difference in female bass (Table 6.10). Liver EROD activity in males is significantly lower in the assessment area and is negatively correlated with liver and fillet PCB concentrations. CYP1A protein expression is not significantly different between the sampling locations. Fish exposed to PCBs generally exhibit EROD induction (elevated EROD activity) (Barron et al., 2000) and CYP1A protein expression (Anderson et al., 2003). However, extended exposure to PCBs has also been associated with low or depressed EROD activity (Besselink et al., 1998). The depressed concentrations of these detoxification enzymes may be associated with negative consequences in fish such as accelerated accumulation of PCBs in tissue and the lessened ability to detoxify other contaminant stressors (Anderson et al., 2003).

SOD is a critical antioxidant enzyme involved in the conversion of superoxide anion to hydrogen peroxide and water, and can be elevated in fish exposed to oxidant stress. Liver and spleen SOD are significantly lower in assessment area bass of both genders relative to reference area bass. Liver and spleen SOD activity are negatively correlated with liver and fillet PCB concentrations, suggesting that PCBs may impair detoxification (Anderson et al., 2003).

Levels of plasma vitellogenin, an egg-yolk precursor protein, are significantly lower in females of equivalent reproductive status in the assessment area than in females in the reference area, and concentrations in pooled females from both sites are negatively correlated with lipid normalized PCBs in the liver (Table 6.10). This result suggests that PCBs may be having an antiestrogenic influence on female bass in the Kalamazoo River.

Analysis of histopathological lesions and parameters also shows statistically significant differences between reference and assessment area bass (Table 6.11). Liver glycogen depletion, which is a common lesion observed in fish under a variety of stressful conditions, is more severe in assessment area fish compared to reference area fish. Macrophage aggregates in the liver, head kidneys, and female ovaries are also more severe in assessment area bass than in reference area bass. These lesions are similarly considered nonspecific indicators of contaminant exposure or other stressors.

Table 6.11. Summary of selected histopathology results of KRE smallmouth bass study, 1995

Parameter	Difference between assessment and reference[a]
Severity of liver glycogen depletion	a > r (p = 0.019)
Severity of macrophage aggregates in liver	a > r (p = 0.017)
Severity of macrophage aggregates in head kidney	a > r (p = 0.006)
Severity of macrophage aggregates in posterior ovary (females)	a > r (p = 0.028)
Frequency of liver neoplasms	a = 1, r = 0 (not sig., p > 0.5)
Frequency of liver foci of cellular alteration	a = 3, r = 0 (not sig., p = 0.224)
Frequency of stomach foci of cellular alteration	a = 1, r = 0 (not sig., p > 0.5)
Severity of foreign body granuloma in liver	a < r (p = 0.025)
Severity of foreign body granuloma in head kidney	a < r (p = 0.002)
Severity of foreign body granuloma in trunk kidney	a < r (p = 0.01)
Severity of eosinophilic granular leukocytes in thyroid	a < r (p = 0.005)

a. a = assessment, r = reference.
Source: Anderson et al., 2003.

Preneoplastic (foci of cellular alteration) and neoplastic lesions are found only in bass from the assessment area, although the incidences are not statistically significant between the assessment and reference areas. Of the 15 fish collected from the assessment area, three bass (two females and one male) had foci of cellular alteration in the liver and one male possessed a large neoplastic helatocellular adenoma in the liver and multiple foci of cellular alteration in the stomach. In the Green Bay study described in the previous section, the incidence of these endpoints (preneoplastic and neoplastic lesions) was also higher in PCB-exposed fish than in reference area fish.

Some histopathological parameters are more severe in reference area bass than in assessment area bass (Table 6.11). Foreign body granulomas in the liver, head kidney, and trunk kidney are more prevalent and severe in the reference area than in the assessment area. The prevalence and severity of eosinophilic granular leukocytes in the thyroid is also significantly higher in reference area bass than in assessment area bass. It is notable that many of the foreign body granulomas observed in reference bass were collocated with parasites.

The types of lesions observed in Kalamazoo River assessment area bass are consistent with those seen in other sites where freshwater fish have been exposed to PCBs (Dey et al., 1993; Schrank et al., 1997; Teh et al., 1997; Barron et al., 2000), and there is laboratory evidence that PCBs act as tumor promoters in fish (Bailey et al., 1987; Hendricks et al., 1990; Fabacher et al., 1991).

This study shows that a suite of organosomatic, biochemical and histopathological indices are spatially and statistically associated with PCB exposure in KRE smallmouth bass. Although cause and effect were not established by this study, the results of this study suggest that smallmouth bass within the assessment area of the Kalamazoo River may be adversely affected by exposure to PCBs. Furthermore, the results of the previous section show that PCB concentrations in KRE smallmouth bass livers are within the range of those in Green Bay walleye that also had adverse histopathological changes. Although this study sampled smallmouth bass from only one assessment location, sediment contamination extends throughout the Portage Creek and Kalamazoo River downstream of PRP facilities. Thus, to the extent that the adverse effects observed in the smallmouth bass collected near D Avenue in Kalamazoo are related to PCB exposure, bass in other locations of the river are most likely similarly affected, and the adverse effects would have been occurring for many years prior to the 1995 study.

6.6 Effects on Benthic Invertebrates

Section 4.3 presents a comparison of PCB concentrations in surficial sediment of the Kalamazoo River and Portage Creek with sediment thresholds developed to predict adverse toxicological effects to benthic invertebrates. The comparison presented in that section shows that PCB concentrations in sediments of the KRE exceed concentrations predicted to cause adverse

impacts to benthic invertebrates, leading to the conclusion that PCB sediment concentrations are sufficient to cause injury to benthic invertebrates.

There are no data available to directly assess the effects of PCBs in the KRE on benthic invertebrates. There are no co-located PCB concentrations and benthic invertebrate community data available, nor are any sediment toxicity tests available. Nevertheless, because surficial sediment PCB concentrations exceed concentrations predicted to cause adverse effects to benthic invertebrates, the Trustees conclude that benthic invertebrates are injured in the Kalamazoo River and Portage Creek.

6.7 Conclusions

Available data on TCDD-eq concentrations from PCBs in KRE smallmouth bass and walleye eggs are inconclusive regarding whether the PCBs are sufficient to cause embryomortality in these fish. The measured TCDD-eq concentrations in the KRE bass and walleye egg samples are less than most effects thresholds. However, the concentrations are greater than the threshold concentration from one study in which rainbow trout were chronically exposed to PCBs prior to egg laying. The sensitivity of walleye and smallmouth bass eggs to embryotoxicity from PCB exposure is not known. Therefore, the available data are inconclusive.

PCB concentrations in smallmouth bass livers suggest that the bass may be injured [per the definition in 43 C.F.R. § 11.62(f)(1)(i)]. Concentrations were generally below literature effects levels, but few studies are available for direct comparison. However, many samples had PCB concentrations in the range of concentrations associated with cancerous tumors and precancerous lesions from a field study in Green Bay, Wisconsin (Barron et al., 2000).

Smallmouth bass collected from the Kalamazoo River assessment area had significant alterations of body condition, endocrine function, and histopathological status compared to those collected in upstream reference areas. The types of biochemical responses and histopathological observations were consistent with those seen in other sites where freshwater fish have been exposed to PCBs. These observations, in combination with the comparison of PCB concentrations in fish livers from the KRE with those from Green Bay, suggest that smallmouth bass (and potentially other species, as well) are exposed to PCBs at concentrations sufficient to cause adverse health effects. To the extent that the alterations observed in the smallmouth bass are associated with PCB exposure, it is likely that smallmouth bass throughout the Kalamazoo River downstream of PRP facilities have been experiencing similar effects from the time of the initial releases. However, the available data do not allow for a determination of injury to be made with a reasonable degree of certainty, and additional studies may be necessary to evaluate and define the scope of injuries. The Trustees conclude that smallmouth bass may be and may have been injured as a result of PCB releases.

Additionally, PCB concentrations in surface sediments are high enough to cause injury to benthic invertebrates. PCB concentrations exceed toxic threshold concentrations for benthic invertebrates by up to two orders of magnitude. Thus, the Trustees conclude that benthic invertebrates are injured throughout the Kalamazoo River and Portage Creek downstream of PRP facilities.

7. Injuries to Wildlife

This chapter presents the Stage I Injury Assessment for wildlife resources of the Kalamazoo River and Portage Creek. The majority of the Kalamazoo River corridor downstream of the city of Kalamazoo is relatively undeveloped. The lower Kalamazoo River is designated a "Natural River" under authority of Michigan's Natural Rivers Act (Part 305 P.A. 451, 1994) (MDNR, 2002). This program is in place to preserve, protect, and enhance Michigan's river systems by maintaining these rivers and adjoining land in as natural a state as possible by preventing unwise use and development. The river's adjoining riparian areas provide essential habitat for many wildlife species because of proximity to the water. Wildlife frequently use travel corridors where vegetation extends along the river and its banks for much of its length (MDNR, 2002).

The Kalamazoo River contains some of the most extensive riparian habitat in southwestern Michigan, providing ample habitat for wildlife (Blasland, Bouck & Lee, 2000c). Sections of the Kalamazoo River corridor, including the Allegan State Game Area and the private Pottawattamie Fish and Game Club, are reserved and managed specifically for wildlife resources. Riparian zones along the Kalamazoo River provide food and cover for both aquatic organisms and terrestrial organisms (Blasland, Bouck & Lee, 2000c).

Some of the riparian areas of the KRE contain high concentrations of PCBs deposited by the river as well as habitat for wildlife. The former impoundments behind the Plainwell, Otsego, and Trowbridge dams contain approximately 510 acres of former sediments as floodplain soils, much of which contains relatively high concentrations of PCBs (Blasland, Bouck & Lee, 1992, 2000b). These former impoundment areas also support wildlife habitat, creating the potential for wildlife to be exposed to PCBs in these areas. For example, the former Trowbridge impoundment contains 374 acres of palustrine forested, emergent, and scrub/shrub wetlands (Figure 7.1).

The riparian wetland habitat of the Kalamazoo River supports many birds, including waterfowl, game birds, raptors, and songbirds (Blasland, Bouck & Lee, 2000c). Extensive marshes, especially downstream of Lake Allegan, provide important resting and feeding habitat for waterfowl, shorebirds, and other birds during migration. Bird surveys were conducted in riparian habitat along the river from Battle Creek to Saugatuck in the spring of 1992 and 1993 by the Kalamazoo River Nature Center, and transects in the Allegan State Game area were resurveyed in 1994 (Adams et al., 1998). Approximately 100 species were observed during each year of the survey (Adams et al., 1998). Species observed in these surveys that use wetland habitat are listed in Table 7.1. A high proportion (about 60%) of birds observed along the Kalamazoo River are neotropical migrants, which breed in the United States or Canada and migrate to Central or South America for winter. Other species use the Kalamazoo River area as winter habitat. Resident species are also present (Adams et al., 1998). It should be noted that the survey methods favored

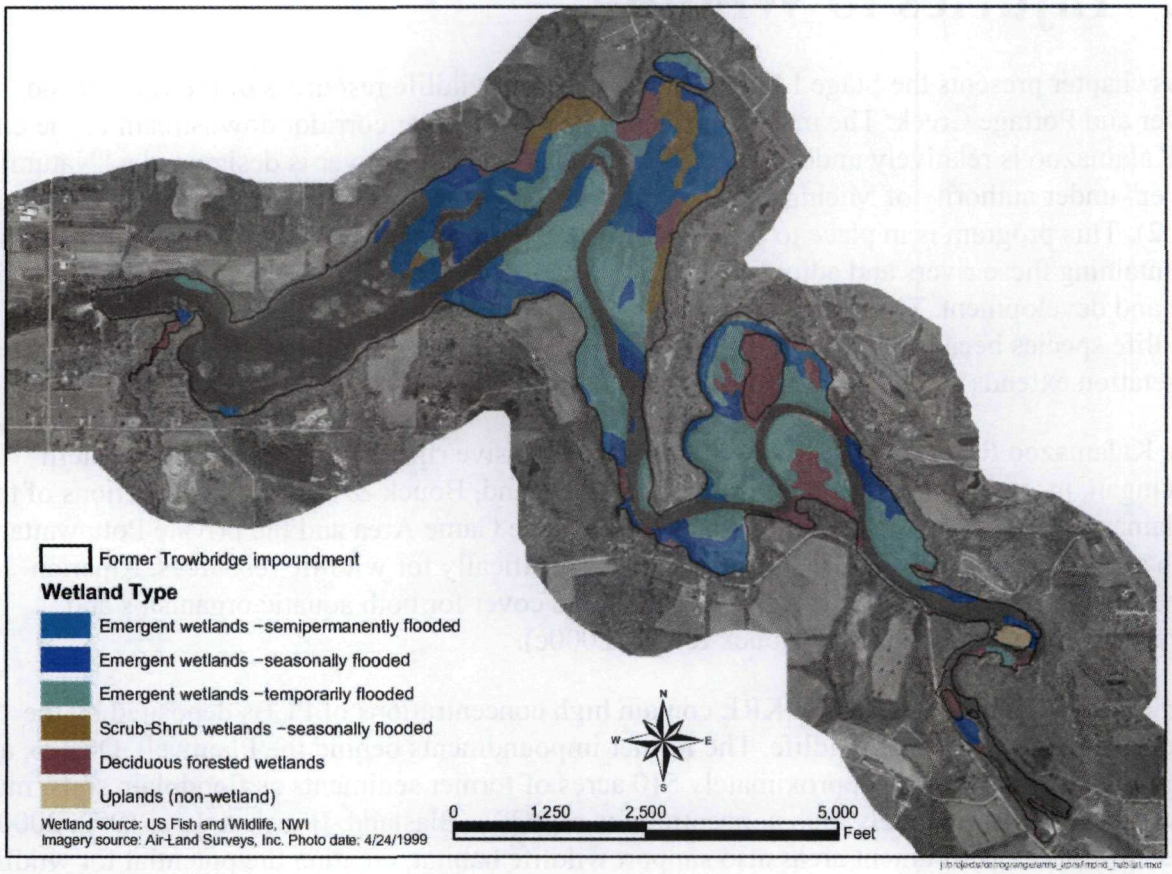

Figure 7.1. Wetland types within the former Trowbridge impoundment.

the observation of birds which were active in more open areas (Adams et al., 1998). Many species of birds in addition to those presented in Table 7.1 are expected to be found in the Kalamazoo River Basin (Blasland, Bouck & Lee, 2000c).

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status

Scientific name	Common name	Status[a]
Birds		
Podilymbus podiceps	Pied-billed grebe	
Ardea herodias	Great blue heron	
Ardea alba	Great egret	
Butorides virescens	Green heron	
Nycticorax nycticorax	Black-crowned night-heron	MI - SC
Cathartes aura	Turkey vulture	
Branta canadensis	Canada goose	
Cygnus olor	Mute swan	
Aix sponsa	Wood duck	
Anas rubripes	American black duck	
Anas platyrhynchos	Mallard	
Anas discors	Blue-winged teal	
Bucephala clangula	Common goldeneye	
Lophodytes cucullatus	Hooded merganser	
Pandion haliaetus	Osprey	MI - T
Haliaeetus leucocephalus	Bald eagle	US - T
Circus cyaneus	Northern harrier	MI - SC
Accipiter striatus	Sharp-shinned hawk	
Buteo jamaicensis	Red-tailed hawk	
Falco sparverius	American kestrel	
Phasianus colchicus	Ring-necked pheasant	
Bonasa umbellus	Ruffed grouse	
Meleagris gallopavo	Wild turkey	
Grus canadensis	Sandhill crane	
Charadrius vociferus	Killdeer	
Actitis macularia	Spotted sandpiper	
Scolopax minor	American woodcock	
Larus delawarensis	Ring-billed gull	
Chlidonias niger	Black tern	MI - SC
Columba livia	Rock dove	
Zenaida macroura	Mourning dove	
Coccyzus erythropthalmus	Black-billed cuckoo	
Coccyzus americanus	Yellow-billed cuckoo	
Otus asio	Eastern screech-owl	

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status (cont.)

Scientific name	Common name	Status[a]
Bubo virginianus	Great horned owl	
Strix varia	Barred owl	
Chaetura pelagica	Chimney swift	
Archilochus colubris	Ruby-throated hummingbird	
Ceryle alcyon	Belted kingfisher	
Melanerpes erythrocephalus	Red-headed woodpecker	
Melanerpes carolinus	Red-bellied woodpecker	
Sphyrapicus varius	Yellow-bellied sapsucker	
Picoides pubescens	Downy woodpecker	
Picoides villosus	Hairy woodpecker	
Colaptes auratus	Northern flicker	
Dryocopus pileatus	Pileated woodpecker	
Contopus virens	Eastern wood-pewee	
Empidonax virescens	Acadian flycatcher	
Empidonax traillii	Willow flycatcher	
Empidonax minimus	Least flycatcher	
Sayornis phoebe	Eastern phoebe	
Myiarchus crinitus	Great crested flycatcher	
Tyrannus tyrannus	Eastern kingbird	
Vireo flavifrons	Yellow-throated vireo	
Vireo solitarius	Blue-headed (solitary) vireo	
Vireo gilvus	Warbling vireo	
Vireo olivaceus	Red-eyed vireo	
Cyanocitta cristata	Blue jay	
Corvus brachyrhynchos	American crow	
Progne subis	Purple martin	
Tachycineta bicolor	Tree swallow	
Stelgidopteryx serripennis	Northern rough-winged swallow	
Riparia riparia	Bank swallow	
Petrochelidon pyrrhonota	Cliff swallow	
Hirundo rustica	Barn swallow	
Poecile atricapilla	Black-capped chickadee	
Baeolophus bicolor	Tufted titmouse	
Sitta carolinensis	White-breasted nuthatch	
Certhia americana	Brown creeper	

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status (cont.)

Scientific name	Common name	Status[a]
Thryothorus ludovicianus	Carolina wren	
Troglodytes aedon	House wren	
Cistothorus palustris	Marsh wren	MI - SC
Polioptila caerulea	Blue-gray gnatcatcher	
Sialia sialis	Eastern bluebird	
Catharus fuscescens	Veery	
Hylocichla mustelina	Wood thrush	
Turdus migratorius	American robin	
Dumetella carolinensis	Gray catbird	
Toxostoma rufum	Brown thrasher	
Sturnus vulgaris	European starling	
Bombycilla cedrorum	Cedar waxwing	
Vermivora pinus	Blue-winged warbler	
Vermivora peregrina	Tennessee warbler	
Parula americana	Northern parula warbler	
Dendroica petechia	Yellow warbler	
Dendroica pensylvanica	Chestnut-sided warbler	
Dendroica caerulescens	Black-throated blue warbler	
Dendroica virens	Black-throated green warbler	
Dendroica fusca	Blackburnian warbler	
Dendroica cerulea	Cerulean warbler	MI - SC
Setophaga ruticilla	American redstart	
Protonotaria citrea	Prothonotary warbler	MI - SC
Seiurus aurocapillus	Ovenbird	
Seiurus motacilla	Louisiana waterthrush	MI - SC
Oporornis philadelphia	Mourning warbler	
Geothlypis trichas	Common yellowthroat	
Piranga olivacea	Scarlet tanager	
Pipilo erythrophthalmus	Eastern towhee	
Spizella passerina	Chipping sparrow	
Spizella pusilla	Field sparrow	
Melospiza melodia	Song sparrow	
Melospiza georgiana	Swamp sparrow	
Cardinalis cardinalis	Northern cardinal	
Pheucticus ludovicianus	Rose-breasted grosbeak	

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status (cont.)

Scientific name	Common name	Status[a]
Passerina cyanea	Indigo bunting	
Agelaius phoeniceus	Red-winged blackbird	
Sturnella magna	Eastern meadowlark	
Quiscalus quiscula	Common grackle	
Molothrus ater	Brown-headed cowbird	
Icterus galbula	Baltimore oriole	
Carpodacus mexicanus	House finch	
Carduelis tristis	American goldfinch	
Coccothraustes vespertinus	Evening grosbeak	
Passer domesticus	House sparrow	
Amphibians		
Acris crepitans blanchardi	Blanchard's cricket frog	MI - SC
Ambystoma laterale	Blue-spotted salamander	
Ambystoma maculatum	Spotted salamander	
Ambystoma opacum	Marbled salamander	MI - T
Ambystoma tigrinum	Tiger salamander	
Bufo americanus	American toad	
Bufo fowleri	Fowler's toad	
Hemidactylium scutatum	Four-toed salamander	
Hyla versicolor	Gray treefrog	
Necturus maculosus	Mudpuppy	
Notophthalmus viridescens	Eastern newt	
Plethodon cinereus	Eastern red-backed salamander	
Pseudacris crucifer	Spring peeper	
Pseudacris triseriata	Western chorus frog	
Rana catesbeiana	American bullfrog	
Rana clamitans	Green frog	
Rana palustris	Pickerel frog	
Rana pipiens	Northern leopard frog	
Rana sylvatica	Wood frog	
Reptiles		
Apalone spinifera	Eastern spiny softshell	
Chelydra serpentina	Snapping turtle	
Chrysemys picta	Painted turtle	
Clemmys guttata	Spotted turtle	MI - T

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status (cont.)

Scientific name	Common name	Status[a]
Clonophis kirtlandii	Kirtland's snake	MI - E
Coluber constrictor foxii	Blue racer	
Diadophis punctatus edwardi	Northern ringneck snake	
Elaphe obsoleta obsoleta	Black rat snake	MI - SC
Emydoidea blandingii	Blanding's turtle	MI - SC
Graptemys geographica	Map turtle	
Eumeces fasciatus	Five-lined skink	
Heterodon platirhinos	Eastern hognose snake	
Lampropeltis trianguium traingulum	Eastern milk snake	
Nerodia sipedon sipedon	Northern water snake	
Opheodrys vernalis	Smooth green snake	
Regina septemvittata	Queen snake	
Sistrurus catenatus catenatus	Eastern massasauga rattlesnake	MI - SC; US- C
Sternotherus odoratus	Musk turtle (stinkpot)	
Storeria dekayi	Brown snake	
Storeria occipitomaculata occipitomaculata	Northern red-bellied snake	
Terrapene carolina carolina	Eastern box turtle	MI - SC
Thamnophis butleri	Butler's garter snake	
Thamnophis sauritus septentrionalis	Northern ribbon snake	
Thamnophis sirtalis sirtalis	Eastern garter snake	
Mammals		
Blarina brevicauda	Shorttail shrew	
Canis latrans	Coyote	
Castor canadensis	Beaver	
Condylura cristata	Starnose mole	
Cryptotis parva	Least shrew	MI - T
Didelphis marsupialis	Opossum	
Eptesicus fuscus	Big brown bat	
Erethizon dorsatum	Porcupine	
Felis rufus	Bobcat	
Glaucomys volans	Southern flying squirrel	
Lasionycteris noctivagans	Silver-haired bat	
Lasiurus borealis	Red bat	
Lasiurus cinereus	Hoary bat	

Table 7.1. Wildlife species observed in the Kalamazoo River Basin that utilize wetland habitat, and their protection status (cont.)

Scientific name	Common name	Status[a]
Lutra canadensis	River otter	
Marmota monax	Woodchuck	
Mephitis mephitis	Striped skunk	
Microtus pinetorum	Woodland vole	MI - SC
Microtus ochrogaster	Prairie vole	MI - E
Microtus pennsylvanicus	Meadow vole	
Mus musculus	House mouse	
Mustela erminea	Ermine	
Mustela frenata	Longtail weasel	
Mustela nivalis	Least weasel	
Mustela vison	Mink	
Myotis keenii	Keen's bat	
Myotis lucifugus	Little brown bat	
Nycticeius humeralis	Evening bat	
Odocoileus virginianus	Whitetail deer	
Ondatra zibethicus	Muskrat	
Peromyscus leucopus	White-footed mouse	
Peromyscus maniculatus	Deer mouse	
Procyon lotor	Raccoon	
Scalopus aquaticus	Eastern mole	
Sciurus carolinensis	Eastern gray squirrel	
Sciurus niger	Eastern fox squirrel	
Sorex cinereus	Masked shrew	
Spermophilus tridecemlineatus	Thirteen-lined ground squirrel	
Sylvilagus floridanus	Eastern cottontail	
Synaptomys cooperi	Southern bog lemming	
Tamias striatus	Eastern chipmunk	
Tamiasciurus hudsonicus	Red squirrel	
Taxidea taxus	Badger	
Urocyon cinereoargenteus	Gray fox	
Vulpes vulpes	Red fox	
Zapus hudsonius	Meadow jumping mouse	

a. State listings (MI) from Michigan Natural Features Inventory (2002); Federal (U.S.) from U.S. FWS (2003b). E = endangered, T = threatened, SC = special concern, C = under consideration for listing.

Source: Birds from Adams et al. (1998); other animals from Blasland, Bouck & Lee (2000c).

Nine avian species identified as endangered, threatened or of special concern were observed during the surveys: black-crowned night heron, black tern, bald eagle, northern harrier, osprey, marsh wren, prothonotary warbler, cerulean warbler, and Louisiana waterthrush (Adams et al., 1998; Michigan Natural Features Inventory, 2002; U.S. FWS, 2003a).

Reptilian, amphibian, and mammalian species that utilize riparian habitat in the KRE are also listed in Table 7.1. These species have been observed in the Allegan State Game Area (MDNR, 1993b, as cited in Blasland, Bouck & Lee, 2000c). Several species are identified as endangered, threatened or of special concern, including Blanchard's cricket frog, marbled salamander, spotted turtle, Kirtland's snake, black rat snake, Blanding's turtle, eastern massasauga rattlesnake, eastern box turtle, least shrew, woodland vole and prairie vole.

The Indiana bat (*Myotis sodalis*) is listed as endangered by both the state and federal governments and may be present in the KRE even though it was not reported as observed in the KRE by Blasland, Bouck & Lee (2000c). The U.S. FWS considers that Indiana bats may be present in suitable habitat throughout the southern three tiers of counties in Michigan. Suitable summer habitat for the Indiana bat consists of floodplain and upland forests with roost trees that have exfoliating bark. The KRE contains such habitat and is within the known range of the Indiana bat, so the bat may be present and detectable using species-specific survey methods.

Ecosystem services provided by wildlife include prey for carnivorous and omnivorous wildlife, control of prey populations, and nutrient and energy cycling. Human use services include various types of recreation (hunting, birdwatching) and as supplemental food sources.

7.1 Injury Definitions

Biological resources are defined in the DOI regulations as "those natural resources referred to in section 101(16) of CERCLA as fish and wildlife and other biota. Fish and wildlife include marine and freshwater aquatic and terrestrial species; game, nongame, and commercial species; and threatened, endangered, and state sensitive species. Other biota encompass shellfish, terrestrial and aquatic plants, and other living organisms not listed in this definition" [43 C.F.R. § 11.14(f)]. This chapter addresses injuries to wildlife; injuries to aquatic biota were addressed separately in Chapters 5 and 6 of this document.

According to DOI regulations, "an injury to a biological resource has resulted from the . . . release of a hazardous substance if concentration of the substance is sufficient to" [43 C.F.R. § 11.62(f)(1)]:

- Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 C.F.R. § 11.62 (f)(1)(ii)]

- Cause the biological resource or its offspring to have undergone at least one of the following adverse changes in viability: death, disease, behavioral abnormalities, cancer, genetic mutations, physiological malfunctions (including malfunctions in reproduction), or physical deformations [43 C.F.R. § 11.62(f)(1)(i)].

An injury to biological resources can be demonstrated, per the DOI regulations, "if the biological response under consideration can satisfy all of the following acceptance criteria" [43 C.F.R. § 11.62 (f)(2)]: (i-iv)]:

- The biological response is often the result of exposure to . . . [the] hazardous substances [43 C.F.R. § 11.62 (f)(2)(i)].

- Exposure to . . . [the] hazardous substances is known to cause this biological response in free-ranging organisms [43 C.F.R. § 11.62 (f)(2)(ii)].

- Exposure to . . . [the] hazardous substances is known to cause this biological response in controlled experiments [43 C.F.R. § 11.62 (f)(2)(iii)].

- The biological response measurement is practical to perform and produces scientifically valid results [43 C.F.R. § 11.62 (f)(2)(iv)].

Injuries to biological resources may include death [43 C.F.R. § 11.62 (f)(4)(i)], as confirmed by laboratory toxicity testing [43 C.F.R. § 11.62 (f)(4)(i)(E)], behavioral abnormalities [43 C.F.R. § 11.62 (f)(4)(iii)(B)], and physiological malfunctions [43 C.F.R. § 11.62 (f)(4)(v)].

7.2 Stage I Injury Assessment Approach

Exposure to PCBs is known to cause a wide range of adverse effects in birds (outlined in Table 7.2). PCB exposure can cause death in avian embryos and juvenile and adult birds as well as sublethal and reproductive effects. Female reproductive systems in birds are generally the most sensitive endpoints to PCB toxicity (Peterson et al., 1993; Safe, 1994). PCBs have been found to cause chromosome alteration and to increase susceptibility to disease. Behavioral abnormalities such as impaired courtship, abnormal nest building behavior, and impaired avoidance behavior have also been observed in birds exposed to PCBs. Physiological impairments caused by PCB exposure in birds include decreased fecundity and altered biochemistry. Physical deformations such as beak and skeletal deformities, increased liver weights, and histopathological lesions are also known effects of PCB exposure in avian species.

Table 7.2. Overview of adverse effects in birds caused by exposure to PCBs

Category	Response measure	Documented response	Example studies
Death	Mortality	Increased adult and juvenile mortality	Heath et al., 1972; Stickel et al., 1984
		Increased embryomortality	Carlson and Duby, 1973; Brunström and Reutergårdh, 1986
Cancer/genetic mutation	Genetic mutation	Chromosome alteration	Peakall et al., 1972
Disease	Immune system impairment	Increased susceptibility to viral challenge	Friend and Trainer, 1970
Behavioral abnormalities	Reproductive behavior impairment	Reduced parental incubation attentiveness	Peakall and Peakall, 1973
		Impaired courtship behavior	Tori and Peterle, 1983
Behavioral abnormalities	Behavioral impairment	Abnormal nest building behavior	McCarty and Secord, 1999
		Impaired avoidance of visual cliff	Dahlgren and Linder, 1971
Physiological malfunction	Reproductive impairment	Reduced fecundity	Lincer and Peakall, 1970; Peakall et al., 1972; Carlson and Duby, 1973; Brunström and Reutergårdh, 1986
		Eggshell thinning	Haseltine and Prouty, 1980
Physiological malfunction	Biochemical changes	Reduced estrogen levels	Chen et al., 1994
		Porphyria	Elliott et al., 1990
		Altered vitamin A status	Spear et al., 1989
		Enzyme induction	Brunström and Lund, 1988
Physical deformation	Deformities	External malformation (e.g., small beak, eyes, unabsorbed yolk sac)	Brunström and Lund, 1988; Hoffman et al., 1998
		Skeletal deformities	Hoffman et al., 1998
		Increased heart weights	Hansen et al., 1976; Heid et al., 2001
		Increased liver weights	Elliott et al., 1997
		Histopathological liver lesions	Hoffman et al., 1996b

Mammalian exposure to PCBs has been found to cause similar types of effects (Table 7.3). As with birds, female reproductive systems are generally the most sensitive endpoints to PCB toxicity. Exposure to PCBs can result in mortality, sublethal effects, and reproductive impairment in mammals. Behavioral abnormalities, such as learning deficits, have been observed in rats exposed to PCBs via maternal exposure. Physiological impairment effects include reduced reproductive success and offspring mortality as well as altered vitamin status and enzyme induction. PCB exposure has also been known to cause physical deformities such as jaw deformities, increased organ weights, and deformed nail growth in mink. Mink are particularly sensitive to PCBs and have been studied extensively.

Table 7.3. Overview of adverse effects in mammals caused by exposure to PCBs

Category	Response measure	Documented response	Example studies
Death	Mortality	Increased mortality	Aulerich et al., 1973, 1985; Platonow and Karstad, 1973; Aulerich and Ringer, 1977; Bleavins et al., 1980
Behavioral abnormalities	Neurological impairment	Neurodevelopmental deficits	Lilienthal and Winneke, 1991
Behavioral abnormalities	Dietary impairment	Refusal to eat/anorexia	Aulerich et al., 1985, 1987; Ringer, 1983
Behavioral abnormalities	Reproductive impairment	Decreased sexual receptivity	Brezner et al., 1984
		Delayed copulation	Brezner et al., 1984
Physiological malfunction	Reproductive impairment	Reduced reproductive success	Platonow and Karstad, 1973; Aulerich et al., 1985; Bäcklin and Bergman, 1992; Restum et al., 1998
		Mortality of offspring	Hornshaw et al., 1983; Ringer, 1983; Brezner et al., 1984; Aulerich et al., 1985; Wren et al., 1987b; Heaton et al., 1995a; Restum et al., 1998
		Reduced weight gain in offspring	Brezner et al., 1984; Wren et al., 1987b
Physiological malfunction	Biochemical changes	Altered vitamin A status	Brunström et al., 1991; Håkansson et al., 1992; Käkelä et al., 1999
		Enzyme induction	Aulerich et al., 1985; Brunström et al., 1991; Dragnev et al., 1994; Shipp et al., 1998
		Immunosuppression	Brunström et al., 1991

Table 7.3. Overview of adverse effects in mammals caused by exposure to PCBs (cont.)

Category	Response measure	Documented response	Example studies
Physical deformation	Deformities	Increased organ weights	Hornshaw et al., 1983; Ringer, 1983; Aulerich et al., 1985, 1987; Kihlström et al., 1992; Heaton et al., 1995b; Restum et al., 1998
		Decreased heart weight	Aulerich et al., 1985
		Deformed nail growth	Bleavins et al., 1982; Aulerich et al., 1987
		Jaw deformities (loose teeth, bone loss)	Render et al., 2000
		Weight loss	Brezner et al., 1984; Aulerich et al., 1985, 1987
		Abnormal molting	Aulerich et al., 1987
		Gastric ulcers/internal bleeding	Aulerich et al., 1985, 1987

Little is known about the impact of PCBs and other contaminants on amphibians and reptiles as most studies have focused on reporting contaminant residues, rather than evaluating toxicity (Portelli and Bishop, 2000; Glennemeier and Begnoche, 2002). Bishop et al. (1991) report decreased hatching rates and increased deformity rates in snapping turtle (*Chelydra serpentina*) eggs collected from locations along Lake Ontario which are contaminated with PCBs, dioxins, furans, and pesticides, relative to eggs collected from a control site. Laboratory results indicate that hatching success of amphibians is negatively affected by PCB exposure (Glennemeier and Begnoche, 2002). Field observations in wetlands along the Kalamazoo River showed that densities of amphibian larvae and adults decreased with increased total PCBs in sediments, although this trend was not statistically significant (Glennemeier and Begnoche, 2002). PCB concentrations measured in green frogs (*Rana clamitans*) collected from the Kalamazoo River suggest that amphibians do not concentrate PCBs as highly as other taxa like birds or fish (Glennemeier and Begnoche, 2002). Because the toxicological literature for reptiles and amphibians is not as complete as it is for birds and mammals, potential injury to these species is not evaluated in this Stage I Assessment.

Table 7.4 outlines the approaches taken in this chapter to assess injury to birds and mammals. Concentrations of PCBs in edible portions of mallards are compared to appropriate regulatory standards for human consumption. The results of the ERA conducted by MDEQ are reviewed and PCB concentrations in whole fish are compared to dietary toxicity thresholds for piscivorous birds and mammals. Bald eagle reproductive success is evaluated and measured PCB and dichlorodiphenyl dichloroethylene (DDE) concentrations in bald eagle eggs and plasma are

Table 7.4. Approaches to evaluate injury to wildlife

Injury definition	Stage I injury assessment approach	Chapter section
Exceed action or tolerance levels established under section 402 of the Food, Drug and Cosmetic Act, 21 U.S.C. 342, in edible portions of organisms [43 C.F.R. § 11.62 (f)(1)(ii)].	Compare concentrations of total PCBs in mallard duck breast tissue to FDA action levels for consumption.	7.3
Cause the biological resource or its offspring to have undergone adverse changes in viability [43 C.F.R. § 11.62(f)(1)(i)].	Review MDEQ ERA and compare measured PCB concentrations in whole fish to bald eagle dietary toxicological benchmarks.	7.4
	Evaluate reproductive success of bald eagles in the KRE; compare measured PCB and DDE concentrations in eggs to toxicological benchmarks; compare measured concentrations in nestling plasma to concentrations in other locations in Michigan.	7.5
	Compare concentrations of measured PCBs in eggs of other birds to toxicological benchmarks.	7.6
	Review MDEQ ERA and compare measured PCB concentrations in whole fish to mammalian toxicological benchmarks.	7.7
	Evaluate mink viability by reviewing mink trapping success rates and comparing measured PCB concentrations in mink tissue to toxicological benchmarks.	7.8
	Compare measured PCB concentrations in small mammal, shrew, and muskrat tissue to toxicological benchmarks.	7.9
	Compare floodplain soil PCB concentrations to modeled soil toxicity thresholds.	7.10

compared to reproductive toxicological benchmarks. PCB concentrations in other avian species also are compared to appropriate toxicological benchmarks. Mink trapping rates are evaluated and mink, small mammal, and muskrat tissue concentrations are compared to toxicological benchmarks. Finally, the extensive available data on PCB concentrations in floodplain soils is compared to thresholds for effects to wildlife through bioaccumulation of PCBs into the food chain.

7.3 Mallard Duck Breast Tissue Concentrations

7.3.1 Data sources

PCB concentrations in breast tissue of mallard ducks collected in 1988 (U.S. FWS, 1989; Michigan Department of Public Health, 1990) are used to evaluate injury to wildlife in this section.

The U.S. FWS collected one mallard duck from along the Kalamazoo River in Allegan County, four from Pottawattamie Marsh, and one from Ottawa Marsh. For all mallards except one from Pottawattamie Marsh, both a skin-on and a skin-off breast sample were analyzed at the Mississippi State University Chemical Laboratory for total PCBs on a lipid normalized basis (Mississippi State University, 1990). One mallard from Pottawattamie Marsh only had a skin-on sample collected. A skin-on breast split sample from the mallard collected along the Kalamazoo River (1A) and a skin-off breast split sample from one of the mallards collected from Pottawattamie Marsh (4B) were sent to the Michigan Department of Public Health for a Quality Assurance evaluation (Michigan Department of Public Health, 1990). Four split samples were also analyzed by the Patuxent Wildlife Research Center, including the skin-on and -off portions of the mallard from along the Kalamazoo River (1A & 1B), and the skin-on and -off portions of one mallard from Pottawattamie Marsh (4A & 4B). Although PCB data are available for other waterfowl samples collected in the KRE (MDNR, 1987b), these data were not used in this evaluation because the samples were not of an edible portion, as the DOI regulations require [43 C.F.R. § 11.62 (f)(1)(ii)]. Furthermore, lipid data were not available for these samples for use in deriving estimated concentrations in edible tissues.

7.3.2 Regulatory criteria and standards

Waterfowl resources are injured if they contain concentrations of a hazardous substance sufficient to exceed action levels or tolerances established by the FDA [43 C.F.R. § 11.62 (f)(1)(ii)]. The FDA established a temporary tolerance level of 3 mg/kg ("fat basis") in poultry [21 C.F.R. § 109.30 (a)(3)]. For the purposes of the Stage I Assessment, the Trustees assume that fat basis is equivalent to a lipid-normalized PCB concentration. This level may also be applied to edible portions of wildlife hunted recreationally, such as ducks or geese. However it should be noted that people generally consume less game than commercial poultry, and this tolerance level may be overprotective.

7.3.3 Results

The PCB concentration in the skin-on breast sample from the mallard collected from along the Kalamazoo River in Allegan County (1A), 7.7 mg/kg on a lipid basis, was greater than the FDA tolerance level of 3 mg/kg (Table 7.5; Mississippi State University, 1990). However, the split sample of 1A analyzed by Michigan Department of Public Health (1990) had a reported PCB concentration of 1.8 mg/kg lipid, and PCBs were not detected at a limit of 1.0 mg/kg lipid in the split sample analyzed at Patuxent Wildlife Research Center (U.S. FWS, 1989). PCB concentrations were below the analytical detection limit (0.05 mg/kg ww) in the mallards collected from Pottawattamie and Ottawa Marsh (Mississippi State University, 1990). However, the Michigan Department of Public Health (1990) reported a PCB concentration of 1.2 mg/kg lipid in a skin-off split sample from Potawattamie Marsh (4B), and Patuxent Wildlife Research Center reported a PCB concentration of 0.8 mg/kg lipid in a skin-on split sample from Potawattamie Marsh (4A) (U.S. FWS, 1989). Due to the limited number of samples and the inconclusiveness of the analytical data, the Trustees cannot reach any conclusions about injury to waterfowl by exceedences of FDA tolerance levels.

Table 7.5. Total PCB concentrations in breast tissue of mallard ducks, 1988

Sample ID	Sample location	Sample type	Total PCB (mg/kg ww)	Total PCB (mg/kg lipid)	Analyzed by[a]
1A	Kalamazoo River, Allegan County	Skin-on	0.29	7.7	Mississippi State
1A-split	Kalamazoo River, Allegan County	Skin-on	0.079	1.8	MDPH
1A-split	Kalamazoo River, Allegan County	Skin-on	ND (0.049)	ND (1.0)	Patuxent
1B	Kalamazoo River, Allegan County	Skin-off	ND (0.05)	ND (2.9)	Mississippi State
1B-split	Kalamazoo River, Allegan County	Skin-off	ND (0.10)	ND (1.4)	Patuxent
4A	Pottawattamie Marsh	Skin-on	ND (0.05)	ND (0.6)	Mississippi State
4A-split	Pottawattamie Marsh	Skin-on	0.05	0.8	Patuxent
4B	Pottawattamie Marsh	Skin-off	ND (0.05)	ND (2.1)	Mississippi State
4B-split	Pottawattamie Marsh	Skin-off	0.025	1.2	MDPH
4B-split	Pottawattamie Marsh	Skin-off	ND (0.051)	ND (2.7)	Patuxent
5A	Pottawattamie Marsh	Skin-on	ND (0.05)	—	Mississippi State
5B	Pottawattamie Marsh	Skin-off	ND (0.05)	—	Mississippi State

Table 7.5. Total PCB concentrations in breast tissue of mallard ducks, 1988 (cont.)

Sample ID	Sample location	Sample type	Total PCB (mg/kg ww)	Total PCB (mg/kg lipid)	Analyzed by[a]
6A	Pottawattamie Marsh	Skin-on	ND (0.05)	—	Mississippi State
6B	Pottawattamie Marsh	Skin-off	ND (0.05)	—	Mississippi State
7A	Pottawattamie Marsh	Skin-on	ND (0.05)	—	Mississippi State
8A	Ottawa Marsh	Skin-on	ND (0.05)	—	Mississippi State
8B	Ottawa Marsh	Skin-off	ND (0.05)	—	Mississippi State

a. Mississippi State = Mississippi State University, 1990; MDPH = Michigan Department of Public Health, 1990; Patuxent = U.S. FWS, 1989.

7.4 PCBs in Bird Diet

In this section, the results of MDEQ's ecological risk assessment for PCB exposure to Kalamazoo River area birds are reviewed, and PCB concentrations measured in KRE fish are compared to dietary toxicity thresholds for piscivorous birds.

7.4.1 Data sources

The following data sources are used to evaluate injury to birds in this section:

- Bird dietary exposure model from Kalamazoo River Ecological Risk Assessment (Camp Dresser & McKee, 2003b)
- Data on PCB concentrations in whole fish collected between 1993 and 1999 by Blasland, Bouck & Lee (2001)
- Data on PCB concentrations in whole fish collected in 1999 and 2000 by the MDEQ (Camp Dresser & McKee, 2001, 2002b)
- Data on PCB concentrations in whole fish collected in 1999 by Michigan State University Aquatic Toxicology Laboratory (2002j).

As part of the RI for the Kalamazoo River site, MDEQ conducted an ecological risk assessment that characterizes the risk to wildlife, including birds, from exposure to PCBs in the KRE. MDEQ's ecological risk assessment was reviewed and commented on by EPA, the PRPs, and the public, and the final version (Camp Dresser & McKee, 2003b) incorporates those comments.

Fish represent an important component in the diet of certain bird species, and whole body fish PCB concentrations are used to assess injuries to birds. Blasland, Bouck & Lee (2001) collected 110 whole body samples of golden redhorse (*Moxostoma erythrurum*), northern hogsucker (*Hypentelium nigricans*), spotted sucker (*Minytrema melanops*), and white sucker throughout the KRE in 1993. In 1997, 15 whole body smallmouth bass samples were collected from Plainwell, Lake Allegan, and New Richmond. In 1999, 24 yearling bass were sampled from locations throughout the KRE and 11 white suckers were collected in Portage Creek. All of these samples were analyzed for total PCBs as Aroclors, and these data are used to estimate the dietary exposure of KRE piscivorous birds to PCBs.

The MDEQ collected 36 composite whole body samples of yearling smallmouth bass from eight locations in the Kalamazoo River between the city of Kalamazoo and New Richmond in 1999 and analyzed them for total PCBs (Camp Dresser & McKee, 2001). One composite channel catfish sample was also collected from New Richmond in this year. In 2000, the MDEQ collected five composite whole body samples of yearling white sucker from Portage Creek, one smallmouth bass sample from the Kalamazoo River in the city of Kalamazoo, and five smallmouth bass samples from Lake Allegan (Camp Dresser & McKee, 2002b).

Michigan State University Aquatic Toxicology Laboratory (2002j) collected whole body samples of five carp, four golden redhorse, five smallmouth bass, and one white sucker from Trowbridge in 1999. Additionally, five composite whole body samples of unspecified forage fish were collected from Trowbridge in 1999 (Michigan State University Aquatic Toxicology Laboratory, 2002j).

7.4.2 Toxicity reference value derivation

TRVs can be derived from controlled laboratory toxicity tests where PCBs are administered in bird diets. Dietary toxicity includes the direct effects of ingesting PCB contaminated diets and the effects on offspring when adults ingest PCB contaminated diets. The Trustees compiled and reviewed more than 200 documents to identify studies relevant to the evaluation of dietary effects of PCB concentrations. Studies with a measured dietary dose which were considered reliable are summarized in Tables 7.6-7.9.

Table 7.6. Sublethal effects of PCB exposure to birds

Species	Sex	Life stage	Exposure duration (days)	PCB type	LOEL mg/kg diet (dw)[a]	NOEL mg/kg diet (dw)[a]	Endpoint	Citation
American kestrel	Mixed	Adult	150	Aroclor 1254 Aroclor 1262	0.6		Increased hepatic enzyme activity-oestradiol	Lincer and Peakall, 1970
Ring dove	Unspecified	Adult	56	Aroclor 1254	6.4	0.64	Reduced dopamine concentration; reduced norepinephrine concentration	Heinz et al., 1980
Chicken	Unspecified	Chick	31.5	Aroclor 1248	22	11	Reduced percent hemoglobin	Rehfeld et al., 1972
Chicken	Unspecified	Chick	56	Aroclor 1242 Aroclor 1254	22		Increased liver weight relative to body weight	Hansen et al., 1976
Mallard	Mixed	Chick	10	Aroclor 1254	28		Reduced immunity to duck hepatitis virus (DHV)	Friend and Trainer, 1970
Chicken	Unspecified	Chick	31.5	Aroclor 1248	33	22	Reduced percent blood packed cell volume	Rehfeld et al., 1972
Chicken	Unspecified	Chick	31.5	Aroclor 1248	33	22	Edema	Rehfeld et al., 1971
Chicken	Female	Adult	5	Aroclor 1254	50[b]		Increased hepatic microsomal protein	Chen et al., 1994
Chicken	Mixed	Chick	49	Aroclor 1242	54		Increased zinc absorption	Turk and Heitman, 1976
Mallard	Male	Adult	35	Aroclor 1254	63[b]	12	Increased hepatic EROD/PROD activities; reduced T3 concentration	Fowles et al., 1997
Ring dove	Unspecified	Adult	56	Aroclor 1254	68	6.4	Increased liver weight; reduced hematocrit	Heinz et al., 1980
Chicken	Female	Adult	5	Aroclor 1254	102[b]	52	Increased liver weight relative to body weight; increased hepatic P-450; reduced concentration of estradiol; reduced total plasma calcium	Chen et al., 1994
American kestrel	Female	Adult	28	Aroclor 1254	107[b]		Increased aldrin hepoxidase	Elliott et al., 1997
American kestrel	Female	Adult	84	Aroclor 1254	107[b]		Increased hepatic EROD/APND activities	Elliott et al., 1997

Table 7.6. Sublethal effects of PCB exposure to birds (cont.)

Species	Sex	Life stage	Exposure duration (days)	PCB type	LOEL mg/kg diet (dw)[a]	NOEL mg/kg diet (dw)[a]	Endpoint	Citation
White pelican	Mixed	Adult	70	Aroclor 1254	158		Increased liver weight; increased spleen weight relative to body weight; decreased protein in blood	Greichus et al., 1975
Chicken	Mixed	Chick	49	Aroclor 1242	220	52	Increased calcium absorption	Turk and Heitman, 1976
Chicken	Unspecified	Chick	28	Aroclor 1242	220	110	Hydropericardium	McCune et al., 1962
Chicken	Male	Chick	42	Aroclor 1254	275		Reduced comb weight	Platonow and Funnell, 1971
Chicken	Male	Chick	63	Aroclor 1254	275		Reduced testes weight	Platonow and Funnell, 1971
Mallard	Male	Adult	35	Aroclor 1254	313[b]	63	Increased liver weight relative to body weight; increased hepatic P450; reduced plasma glucose concentration	Fowles et al., 1997
Mallard	Male	Adult	35	Aroclor 1254	313[b]		Increased thyroid weight relative to body weight	Fowles et al., 1997
Chicken	Unspecified	Chick	21	Aroclor 1242	440	220	Abdominal and subcutaneous edema, hydrocardium	Flick et al., 1965
Chicken	Unspecified	Chick	28	Aroclor 1242	440	220	Increased liver weight; enteritis; hemorrhage of internal organs	McCune et al., 1962
Chicken	Male	Chick	60	Aroclor 1260	440		Increased liver weight; reduced spleen weight	Vos and Koeman, 1970
Chicken	Male	Chick	60	Clophen A60	440		Hydropericardium, abdominal and subcutaneous edema; liver necrosis	Vos and Koeman, 1970
Chicken	Unspecified	Chick	28	Aroclor 1242	880	440	Enlarged heart; kidney and liver damage	McCune et al., 1962

a. Where not specified in source document, reported dose was assumed to be on a wet weight basis and was converted to a dry weight basis assuming 10% moisture in commercial feed (see text).
b. Extrapolated diet concentration based on body sizes and feeding rates in the study, or based on sizes and feeding rates in Sample et al. (1996).

Table 7.7. Effects of PCB exposure on egg or sperm production

Species	Sex	Life stage	Study duration (days)	Exposure duration (days)	PCB type	LOEL mg/kg diet (dw)[a]	NOEL mg/kg diet (dw)[a]	Endpoint	Citation
Chicken	Mixed	Adult	273	273	Aroclor 1254	5.5		Reduced egg production	Platonow and Reinhart, 1973
Chicken	Female	Adult	112	63	Aroclor 1232 Aroclor 1242 Aroclor 1248 Aroclor 1254	22	2.2	Reduced egg production	Lillie et al., 1974
Chicken	Female	Adult	56	56	Aroclor 1248	22	11	Reduced egg production	Scott, 1977
American kestrel	Female	Adult	> 365	100	Mixtures of Aroclors	22		Reduced egg production in second generation birds	Fernie et al., 2001
American kestrel	Male	Adult	62	62	Aroclor 1254	100[b]		Reduced sperm concentration, sperm per ejaculate	Bird et al., 1983

a. Where not specified in source document, reported dose was assumed to be on a wet weight basis and was converted to a dry weight basis assuming 10% moisture in commercial feed (see text).
b. Dose was expressed in the study as wet weight in cockerel breast (33 mg/kg). Dose was converted to a dry weight basis assuming 66% moisture in cockerel breast (see text).

Table 7.8. Effects of PCB exposure on avian offspring sublethal endpoints

Species	Life stage	Study duration (days)	Experiment duration (days)	PCB type	LOEL mg/kg diet (dw)[a]	NOEL mg/kg diet (dw)[a]	Endpoint	Citation
Chicken	Chick	112	63	Aroclor 1248 Aroclor 1254	2.2		Reduced growth rate in offspring	Lillie et al., 1974
American kestrel	Egg	180	180	Aroclor 1248	9[b]		Increased shell length and width; reduced shell weight, thickness index, thickness	Lowe and Stendell, 1991
Chicken	Chick	14	14	Aroclor 1254	11		Increased vitamin E-selenium deficiency in progeny	Combs et al., 1975
Chicken	Chick	112	56	Aroclor 1242 Aroclor 1248	11	5.5	Reduced growth rate in offspring	Lillie et al., 1975
Chicken	Embryo	112	63	Aroclor 1232	22		Increased number of embryo abnormalities	Cecil et al., 1974
Chicken	Embryo	112	63	Aroclor 1242 Aroclor 1248 Aroclor 1254	22	2.2	Increased number of embryo abnormalities	Cecil et al., 1974
Chicken	Chick	112	63	Aroclor 1232 Aroclor 1242	22	2.2	Reduced growth rate in offspring	Lillie et al., 1974

a. Where not specified in source document, reported dose was assumed to be on a wet weight basis and was converted to a dry weight basis assuming 10% moisture in commercial feed (see text).
b. Dose was expressed in the study as wet weight in cockerel breast (3 mg/kg). Dose was converted to a dry weight basis assuming 66% moisture in cockerel breast (see text).

Table 7.9. Effects of adult PCB exposure on egg hatchability, young fledged, or egg fertility

Species	Life stage	Study duration (days)	Exposure duration (days)	PCB type	LOEL mg/kg diet (dw)[a]	NOEL mg/kg diet (dw)[a]	Endpoint	Citation
Chicken	Egg	273	273	Aroclor 1254	5.5		Reduced egg fertility	Platonow and Reinhart, 1973
Chicken	Egg	84	42	Aroclor 1242	11	5.5	Reduced number of eggs hatched	Britton and Huston, 1973
Chicken	Egg	112	56	Aroclor 1232 Aroclor 1242 Aroclor 1248	11	5.5	Reduced number of eggs hatched	Lillie et al., 1975
Chicken	Egg	56	56	Aroclor 1248	11	1.1	Reduced number of eggs hatched	Scott, 1977
Chicken	Egg	21	21	Aroclor 1248	11	1.1	Reduced egg hatchability	Scott et al., 1975
Ring dove	Egg	Unknown	Unknown	Aroclor 1254	11		Reduced number of eggs hatched and young fledged	Peakall et al., 1972
Chicken	Egg	42	42	Aroclor 1242	22		Reduced number of eggs hatched	Briggs and Harris, 1973
Chicken	Embryo	98	70	Aroclor 1242 Aroclor 1254	22		Reduced number of eggs hatched	Ax and Hansen, 1975
Chicken	Embryo	112	63	Aroclor 1232	22		Reduced number of eggs hatched	Cecil et al., 1974
Chicken	Embryo	112	63	Aroclor 1242 Aroclor 1248 Aroclor 1254	22	2.2	Reduced number of eggs hatched	Cecil et al., 1974
Chicken	Chick	112	63	Aroclor 1248	22	2.2	Reduced number of eggs hatched	Lillie et al., 1974
Chicken	Egg	112	63	Aroclor 1232 Aroclor 1242 Aroclor 1248 Aroclor 1254	22	2.2	Reduced number of eggs hatched	Lillie et al., 1974
Chicken	Egg	273	98	Aroclor 1254	55	5.5	Reduced number of fertile eggs hatched	Platonow and Reinhart, 1973

a. Where not specified in source document, reported dose was assumed to be on a wet weight basis and was converted to a dry weight basis assuming 10% moisture in commercial feed (see text).

In the documents reviewed, effects concentrations were presented as a dietary concentration (mg PCB/kg dw of food), as an ingested PCB mass (mg PCB/day), or as a body-size-adjusted ingested PCB mass (mg PCB/kg body weight/day). Most data were presented as dietary concentrations (mg PCB/kg dw of food), so where possible and practical, other dietary units were converted to dietary concentrations using body size and ingestion rates (e.g., kilogram food/day) presented in the document. Alternatively, when the study did not present body weight or ingestion rates, representative body sizes and ingestion rates found in Sample et al. (1996) were used. Only PCB dose concentrations expressed as mg/day or mg/kg/day for adult birds were converted to corresponding mg/kg dw of food, since food ingestion rate and body weight can vary considerably for juvenile birds over the course of a study. Most of the studies dosed the birds by adding PCBs to commercial feed mixtures, but in reporting the PCB doses some of the studies did not specify whether the dose was on a dry weight or wet weight basis. For these studies, it was assumed that the reported dose concentration was on a wet weight basis, and a value of 10% moisture content in commercial feed (Peakall and Peakall, 1973) was used to convert to an equivalent dry weight concentration. In addition, two studies (Bird et al., 1983; Lowe and Stendell, 1991) reported PCB doses as mg/kg ww in cockerel breast, the feed used. These concentrations were converted to a dry weight basis assuming 66% moisture in the breast tissue, which is the mean percent moisture reported for waterfowl breast tissue from two studies in New York State (Kim et al., 1984, 1985).

As the studies listed in Table 7.6 show, LOEL concentrations for sublethal endpoints are highly variable, and are largely dependent on the endpoint of concern and the exposure duration, as well as the species used in the study. A 150 day study found increased hepatic enzyme activities in American kestrels at a dietary exposure concentration of 0.6 mg/kg dw in diet (Lincer and Peakall, 1970), whereas a 28 day study on the same species detected enzyme changes at a dietary concentration of 107 mg/kg dw PCB (Elliott et al., 1977). At the high end of the range, enlarged hearts and organ tissue damage were observed in a 28 day study using chicken at 880 mg/kg dw (McCune et al., 1962).

LOEL concentrations for reproductive endpoints are somewhat less variable. Reductions in the number of eggs or sperm produced have been observed in studies of chicken and American kestrel at dietary doses of 5.5 to 100 mg/kg dw (Table 7.7). Sublethal effects in offspring, such as reduced growth rates, abnormalities, and nutritional deficiencies, have been observed at dietary doses of 2.2 to 22 mg/kg dw (Table 7.8). In eight chicken studies and one ring dove study, LOEL concentrations for egg hatchability ranged from 5.5 to 55 mg/kg dw (Table 7.9). In the single study with a dietary LOEL value of 55 mg/kg dw (Platonow and Reinhart, 1973), the next lowest concentration tested was ten times less, suggesting that this value may be considerably higher than a threshold concentration.

Other feeding studies have been conducted which dosed birds with field weathered PCBs (e.g., Summer et al., 1996a; 1996b). These studies fed fish collected from contaminated areas to birds, measured PCB concentrations in the diet, and evaluated a range of toxicological effects. Because these dietary doses potentially contain other contaminants, they are not used in the development of TRVs, but are presented as supporting evidence of field impacts of weathered PCB mixtures. Summer et al. (1996a) found reduced body and liver weights in adult leghorn hens dosed with a diet of fish containing 6.6 mg/kg ww [or 25 mg/kg dw, assuming a moisture content of 74% (Connolly et al., 1992)] PCB. Summer et al. (1996b) found increased embryomortality, decreased hatching rates and various deformities in embryos and chicks of the same adult leghorn hens.

Using the laboratory studies described above and presented in Tables 7.6 to 7.9, TRV ranges can be selected for comparison with PCB concentrations in Kalamazoo River bird diets. TRVs are selected using best professional judgment to capture the central tendency of data for endpoints that appear to be most sensitive to PCB toxicity. They are intended to represent concentrations at which adverse effects are likely to be observed.

For sensitive species such as chicken, embryomortality effects are likely to be observed between 10 and 20 mg/kg PCB dw in bird diet, and sublethal effects are likely to be observed between 0.6 and 2 mg/kg dw, based on the studies listed in Tables 7.6, 7.7, and 7.8. Based on an assumed moisture content of 74% in whole body fish (Connolly et al., 1992), these TRV ranges equate to 2.6 to 5.2 mg/kg for embryomortality and 0.2 to 0.5 mg/kg for sublethal effects on a wet weight basis.

However, these TRVs are based primarily on toxicological data for chickens, and the chicken has consistently been the most sensitive species tested to date for the toxicological effects of PCBs. Therefore, a different set of TRVs is developed for bird species that are less sensitive than chickens to PCB effects. Heath et al. (1972) found that acute LD_{50} concentrations varied across different bird species, including chicken, by a factor of approximately 5. Similarly, Hoffman et al. (1995) concluded that terns, cormorants, and eagles are approximately 5 times less sensitive to PCB effects than chickens. Therefore, for the purposes of this Stage I Assessment, the Trustees use TRVs for more PCB-tolerant species that are five times the TRVs based primarily on data for chickens. Thus, the TRVs for more tolerant bird species is 13 to 26 mg/kg ww for embryomortality and 1.0 to 2.5 mg/kg ww for sublethal effects.

7.4.3 Results

PCB dietary exposure of piscivorous birds

A comparison of measured whole fish PCB concentrations in the Kalamazoo River with dietary TRVs for birds shows that piscivorous birds, such as bald eagles, herons, mergansers, and kingfishers, may be exposed to dietary PCB concentrations that are greater than TRVs. Although fish do not contain PCBs within the embryomortality TRV range for average species, 52 of the 228 fish tissue samples are within or above the range of concentrations that would be expected to cause embryomortality effects in sensitive species (Figure 7.2). Nine golden redhorse collected in 1993, 4 smallmouth bass collected in 1997, and a total of 39 smallmouth bass, carp, golden redhorse, and forage fish composite samples collected in 1999 had total PCB concentrations greater than the lower end of the effects range of 2.6 mg/kg ww.

PCB concentrations in whole fish are within or greater than the TRV range for sublethal effects in piscivorous birds (Figure 7.3). Seventy percent of samples exceed the minimum TRV for more tolerant species, and 95% exceed the minimum TRV for sensitive species. The exceedences of TRVs extend throughout Portage Creek and the mainstem of the Kalamazoo River downstream of PRP facilities. PCB concentrations in whole fish tissue have been elevated since at least 1993 and have continued through at least 2000; however, it is likely that fish have been accumulating PCBs since the time of the initial releases, and also likely that concentrations were much higher at one time than those measured in 1993. Thus it is probable that piscivorous birds such as herons, mergansers, and kingfishers have been exposed to elevated PCB concentrations in their diet since the time of the initial releases.

MDEQ ecological risk assessment

Based on a dietary exposure model and measured concentrations in the KRE, the MDEQ calculated PCB exposure concentrations in the diets of bird species of concern as part of an ecological risk assessment for the RI/FS (Camp Dresser & McKee, 2003b). The avian species evaluated in the ecological risk assessment were the robin, bald eagle, and great horned owl. Using dietary no effect and low effect TRV values derived from the toxicology literature, MDEQ then used a food chain model to estimate the dietary PCB exposure of these bird species. No effect- and low effect-based hazard quotients (HQs) for these species were then calculated as the ratio of the dietary PCB exposure to the TRVs. An HQ of less than one indicates that the species is exposed to a lower concentration in the diet than the no effect or low effect TRV value, while a HQ of greater than one indicates that the species is exposed to a higher concentration. A higher HQ suggests that a species is more at risk to PCB toxicity through dietary exposure.

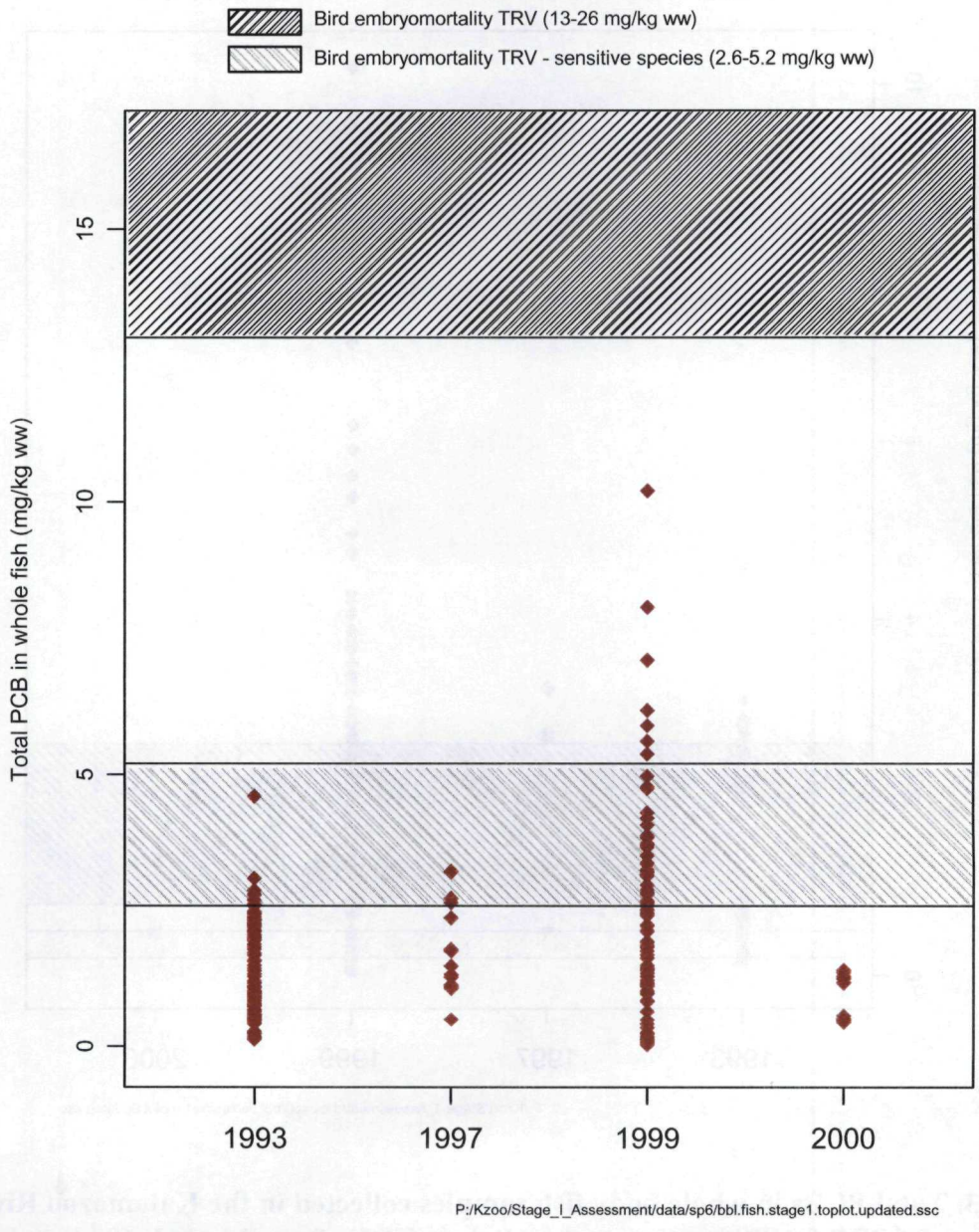

Figure 7.2. Total PCBs in whole body fish samples collected in the Kalamazoo River downstream of PRP facilities compared to dietary TRVs for embryomortality effects in birds. Species include channel catfish, golden redhorse, northern hogsucker, smallmouth bass, spotted sucker, and white sucker.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2001, 2002b; Michigan State University Aquatic Toxicology Laboratory, 2002j.

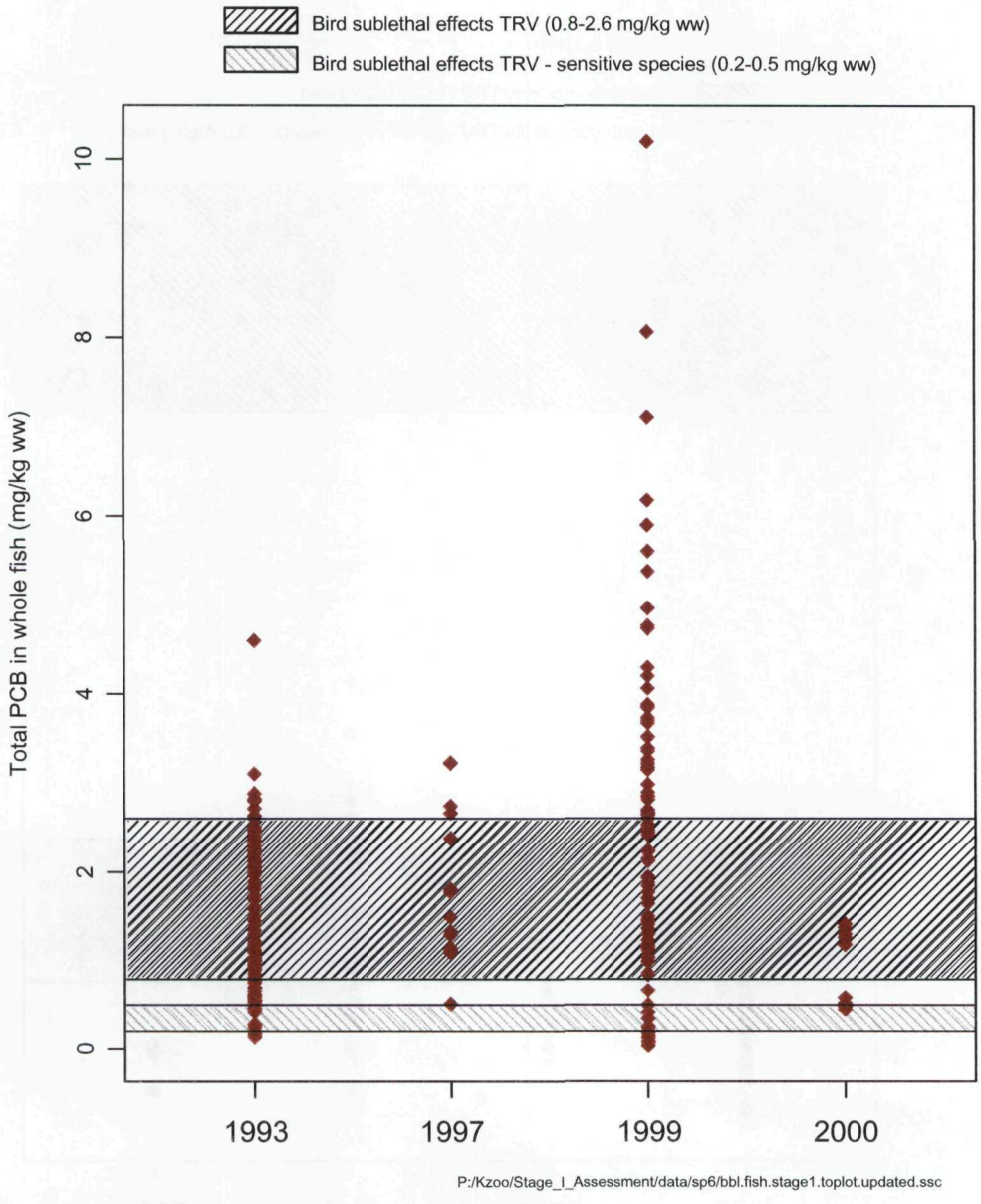

Figure 7.3. Total PCBs in whole body fish samples collected in the Kalamazoo River downstream of PRP facilities compared to dietary TRVs for sublethal effects in birds. Species include channel catfish, golden redhorse, northern hogsucker, smallmouth bass, spotted sucker, and white sucker.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2001, 2002b; Michigan State University Aquatic Toxicology Laboratory, 2002j.

MDEQ concluded that KRE avian receptors are at risk from dietary exposure to PCBs (Table 7.10; Camp Dresser & McKee, 2003b). Piscivorous birds such as bald eagles appear to be at high risk, primarily because of the high concentrations of PCBs in fish. Risks to great horned owl and robin are somewhat lower, however, HQs for these species are greater than 1, and thus are at moderate risk via dietary exposure pathways. However, the ERA indicated that great horned owls may be at greater risk than the dietary HQ indicates, based on measured PCB concentrations in great horned owl eggs collected in the KRE (Camp Dresser & McKee, 2003b; see Section 7.6.3 of this document for an evaluation of PCB concentrations in bird eggs). Studies on great horned owl nesting success and contaminant exposure in the Kalamazoo River area are currently being conducted by the PRPs.

Table 7.10. Summary of MDEQ ERA dietary-based HQ results for birds

Species	No effect TRV-based hazard quotient[a]	Low effect TRV-based hazard quotient[a]
Robin	2.3	1.8
Bald eagle	5.4	4.3
Great horned owl	5.0	1.7

a. Risks are summarized as hazard quotients, which are calculated by dividing the estimated daily exposure concentration by the no effect or low effect TRV. Hazard quotients greater than 1 indicate exposure greater than TRVs.

Source: Camp Dresser & McKee, 2003b.

7.5 Bald Eagle Reproductive Success

7.5.1 Data sources

- Bald eagle nesting observations made by the U.S. FWS (Dave Best, U.S. FWS, personal communication, 2000, 2001, 2002, 2003).
- Data on PCB concentrations in bald eagle eggs collected by Dr. Charles Mehne (2000), the U.S. FWS (Best, 2002), and Michigan State University Aquatic Toxicology Laboratory (2002e).
- Data on PCB concentrations in bald eagle nestling blood plasma collected in 1999 (Summer et al., 2002).

Bald eagle nesting in the Kalamazoo River area and elsewhere in Michigan has been monitored since 1960 (Camp Dresser & McKee, 2001). Bald eagle nesting pairs have been observed annually in the Allegan State Game Area since 1990 (Dave Best, U.S. FWS, personal communication, 2000, 2001). During this time, attempted nests have been inventoried and the number of young successfully reared per occupied nest has been recorded.

Four failed bald eagle eggs were collected from nests in the Allegan State Game Area in 1994 and 1996 and analyzed for total PCBs as Aroclors, the insecticide DDE, and other organochlorine pesticides at the Mississippi State Chemistry Laboratory (Mehne, 2000). An additional bald eagle egg was collected from the same area in 2000 and analyzed for total PCBs and DDE at the U.S. FWS Patuxent Analytical Control Facility (Best, 2002). An additional addled bald eagle egg was collected by Michigan State University Aquatic Toxicology Laboratory (2002e) from Ottawa Marsh in 2000 and analyzed for total PCBs as the sum of congeners.

The MDEQ collected blood plasma from two bald eagle chicks in the Highbanks Game Refuge (Allegan State Game Area) in 1999 (Summer et al., 2002). Plasma samples were analyzed for DDE and for 20 PCB congeners. These congeners include a set of 18 commonly analyzed congeners (8, 18, 28, 44, 52, 66, 101, 105, 118, 128, 138, 153, 170, 180, 187, 195, 206, 209) as well as PCB 110 and PCB 156. The Trustees used a linear regression model to estimate total PCB concentrations from the measured sum of detected congeners.[1]

7.5.2 Benchmarks and injury thresholds

Nesting success

The mean annual bald eagle productivity rate needed to maintain a healthy population has been estimated as 1.0 young per nest (Kubiak and Best, 1991), while a rate of less than 0.7 young per nest is associated with a declining population (Sprunt et al., 1973; Meyer, 1995). Bald eagle nesting success has been observed for several locations in the Great Lakes Region. The productivity rate for all nests in inland Michigan between 1989 and 1993 was 1.0 young per nest

1. The relationship between the sum of 20 measured congeners to total PCBs was developed from eagle and owl plasma PCB congener data from the Kalamazoo River and nearby areas in Michigan. Seventy-seven PCB congeners or co-eluting congener combinations were measured in plasma from 17 nestlings and thus a relationship could be developed from the sum of the same 20 congeners which were measured in other bald eagle plasma samples and the sum of the 77 congeners measured in plasma from these 17 nestlings. The relationship is characterized by the equation: $sum_{77\ congeners}(\mu g/kg) = 4.57 \times sum_{20\ congeners}(\mu g/kg) + 0.98$ (multiple R-squared = 0.9923, residual standard error = 16.659 µg/kg).

(Dykstra et al., 2001), equivalent to the healthy productivity rate postulated by Kubiak and Best (1991).

In the Lower Fox River/Green Bay area, elevated PCBs and DDE have caused a depression in bald eagle productivity (Dykstra et al., 2001). The mean success rate in the Fox River/Green Bay from 1987 to 1996 was 0.55 young per nest.

Egg PCB concentrations

Productivity of bald eagles is not easily evaluated in laboratory experiments, and thus it is difficult to develop unambiguous dose-response relationships for PCBs in bald eagle eggs (Elliott and Harris, 2002). Kubiak and Best (1991), Wiemeyer et al. (1993), Elliott and Harris (2002), and Nisbet and Risebrough (1994) used relationships between geospatial differences in PCB and DDE concentrations and productivity to postulate field-based toxicity thresholds for each contaminant (Table 7.11). Based largely on the Wiemeyer et al. (1993) work, egg toxicity thresholds may be > 3.0 mg/kg ww for PCBs and > 3.6 mg/kg ww for DDE. Major impacts on productivity (reductions of 50% or greater) are possible at PCB concentrations of 13-23 mg/kg ww and DDE concentrations of 3.6-6.3 mg/kg ww. While the studies listed in Table 7.11 found a statistically significant relationship between PCB concentrations in eggs and productivity, Elliott and Harris (2002) did not. Whether individual studies find statistically significant relationships or not appears to depend in part on the degree of PCB contamination being investigated (Elliott and Harris, 2002).

Table 7.11. Bald eagle egg toxicity levels identified from comparisons of regional productivities and contaminant concentrations

Productivity response	Egg PCB toxic level (mg/kg ww)	Egg DDE toxic level (mg/kg ww)	Reference
No productivity reduction		< 2.5	Nisbet and Risebrough, 1994
"Healthy" reproduction	< 1.7	< 6.0	Kubiak and Best, 1991
"Normal" productivity	< 3.0	< 3.6	Wiemeyer et al., 1993
10% productivity reduction	3.0-5.6		Wiemeyer et al., 1993
30% productivity reduction	5.6-13.0		Wiemeyer et al., 1993
30% productivity reduction	—	6 (3.6-12)	Elliott and Harris, 2002
50% productivity reduction	13-23	3.6-6.3	Wiemeyer et al., 1993
Productivity approximately halved		> 5.0	Nisbet and Risebrough, 1994
70% productivity reduction	> 23		Wiemeyer et al., 1993
75% productivity reduction		> 6.3	Wiemeyer et al., 1993

The U.S. FWS and Stratus Consulting (1999) conducted a detailed evaluation of Great Lakes bald eagle nesting success for the Lower Fox River/Green Bay NRDA. Their analysis, which was based on bald eagle nest success and PCB concentration data for nests in Michigan and Wisconsin from 1986 through 1997, concluded that the probability of bald eagle nest success measurably decreases at egg PCB concentrations greater than approximately 20 mg/kg (fresh ww) (Figure 7.4). This threshold concentration is generally consistent with the threshold ranges listed above in Table 7.11 and is reasonably consistent with studies of other species which indicate that thresholds for PCBs in eggs are higher than thresholds for DDE (Elliott and Harris, 2002). Furthermore, in their comprehensive review of the effects of PCBs on bald eagle reproduction, Elliott and Harris (2002) concurred with the 20 mg/kg (fresh ww) value derived in the Lower Fox River/Green Bay NRDA. Therefore, the Stage I Injury Assessment uses a PCB egg concentration of 20 mg/kg (fresh ww) as an injury threshold for bald eagles. This value is close to the upper end of the range (23 mg/kg) from Wiemeyer et al. (1993) for 50% reduction in egg hatching success. The DDE concentration that corresponds to this effect level is 6.3 mg/kg (fresh ww) (Wiemeyer et al., 1993).

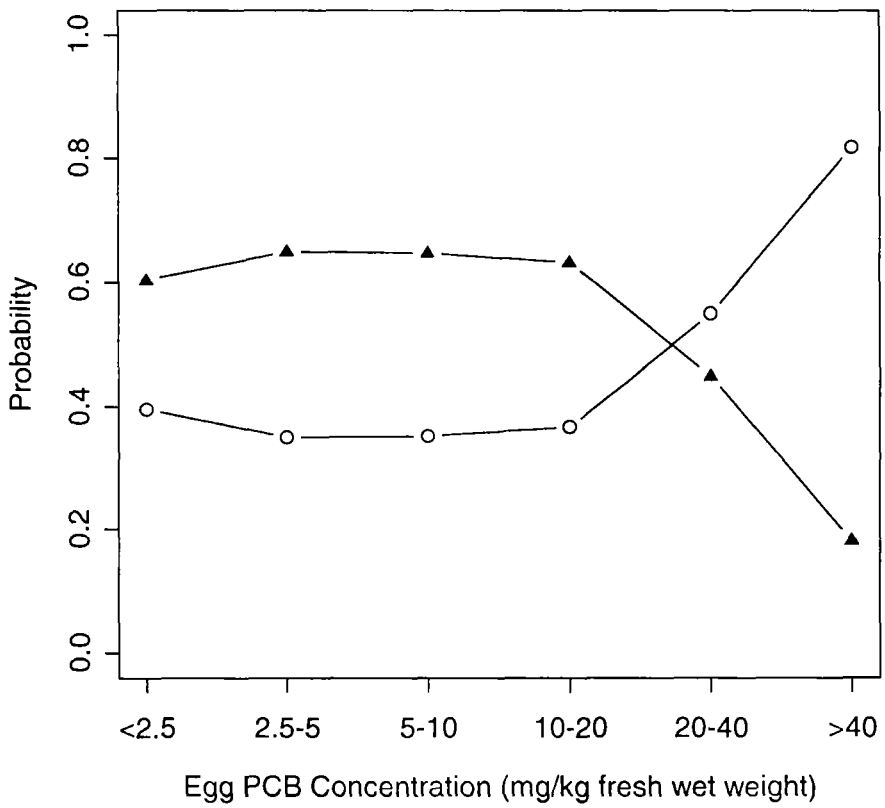

Figure 7.4. Probability of bald eagles in inland Michigan and Wisconsin and Green Bay producing no young (open circles) or one or more young (triangles) in relation to egg PCB concentrations, 1986-1997.

Source: U.S. FWS and Stratus Consulting, 1999.

Nestling plasma concentrations

There are no reliable laboratory TRVs available for the toxicological evaluation of PCB concentrations in bald eagle nestling plasma. However, research has shown that concentrations of both PCBs and DDE in nestling plasma collected in the field are associated with reduced productivity in distinct populations of bald eagles (Bowerman et al., 2003). Nestling plasma PCB concentrations can be used to compare the PCB exposure of Kalamazoo River bald eagle nestlings to the exposure of nestlings in other locations.

Bowerman et al. (2003) measured PCB and DDE concentrations in bald eagle plasma in 10 Great Lakes subpopulations. Plasma samples were collected between 1987 and 1992 and productivity was evaluated from data collected from 1977 to 1992. Geometric mean PCB concentrations in plasma of nestlings from Great Lakes subpopulations were significantly greater ($p = 0.0001$) than those from Voyageurs National Park in Minnesota, or from other interior Michigan or Minnesota subpopulations. Geometric mean DDE concentrations in plasma of nestlings from Great Lakes subpopulations and from Voyageurs National Park were significantly greater ($p = 0.0001$) than those from other interior Michigan or Minnesota subpopulations. Across all the data, productivity was inversely correlated with geometric mean PCB and DDE concentrations in plasma. Based on the regressions of productivity against plasma PCB and DDE concentrations, a productivity rate of 1.0 young per occupied nest was associated with 35 µg/kg PCB and 11 µg/kg DDE, and a productivity rate of 0.7 was associated with 125 µg/kg PCB and 28 µg/kg DDE.

Nestling plasma total PCB concentrations from monitoring programs averaged 546 µg/kg ww from nests along Lake Michigan, 42.6 µg/kg ww in inland Wisconsin, and 18.35 µg/kg ww in the inland Lower Peninsula, Michigan (Dykstra et al., 1998; Summer et al., 2002). The mean concentration of total PCBs in nestling bald eagle plasma in the Lower Fox River and Green Bay, Wisconsin areas was 267 µg/kg ww and 207 µg/kg ww, respectively (Dykstra et al., 1996, 2001). Individual nestling plasma concentrations from the Lower Fox River/Green Bay ranged from 83 to 901 µg/kg ww. The U.S. FWS concluded that bald eagles in this area are exposed to PCBs at concentrations sufficient to cause injury based on reduced productivity (U.S. FWS and Stratus Consulting, 1999).

Because of a general shift from collecting bald eagle eggs to collecting bald eagle plasma, Elliott and Harris (2002) developed a relationship between plasma concentrations of PCBs and DDE using regional mean concentrations for eggs and plasma from the Pacific Northwest and the Great Lakes. Using their relationship for PCBs, a plasma concentration of 189 µg/kg PCB is associated with the threshold of 20 mg/kg ww in eggs described in the previous section of this document. This plasma concentration is slightly higher than the 125 µg/kg PCB in plasma that Bowerman et al. (2003) demonstrated as corresponding to a productivity rate of 0.7 young per occupied nest. A plasma concentration of 44 µg/kg DDE is associated with the threshold of

6.3 mg/kg ww in eggs described in the previous section, which is also higher than the 28 µg/kg DDE threshold that Bowerman et al. (2003) shows corresponding to a productivity rate of 0.7.

7.5.3 Results

Nesting success

From 1960 to 1989, there were no known bald eagle nesting attempts along the Kalamazoo River, with the exception of a single nesting attempt in Ottawa Marsh in 1981. Nesting success for this attempt is unknown. One bald eagle pair has attempted to nest in the Ottawa Marsh since 1990, and a second pair has attempted to nest in the Highbanks Game Refuge (Highbanks Unit/Swan Creek Marsh) since 1993 (Table 7.12; see Figure 1.1). In this period of record, only six young have been successfully reared from these nests, in 1998, 1999, 2000, and 2003. Neither nest was successful in 2001. In 2002, a bald eagle pair attempted to nest in a new area near New Richmond. No eagles were produced in any of the three territories in 2002 (Dave Best, U.S. FWS, personal communication, 2002). In 2003, there were three bald eagle nesting attempts, and one young reared (Dave Best, U.S. FWS, personal communication, 2003).

Table 7.12. Bald eagle productivity in the Kalamazoo River area, 1990-2003

Year	Number of nests attempted	Number of young reared
1990	1	0
1991	1	0
1992	1	0
1993	2	0
1994	2	0
1995	2	0
1996	2	0
1997	2	0
1998	2	2
1999	2	2
2000	2	1
2001	2	0
2002	3	0
2003	3	1

Source: Dave Best, U.S. FWS, personal communication, 2000, 2001, 2002, 2003.

From 1990 through 2003, the mean productivity rate for the three Kalamazoo River bald eagle nests is only 0.2 young per nest attempt, which is a much lower rate than observed in other coastal Lake Michigan nests, inland Michigan nests, or even along the Fox River/Green Bay (Table 7.13). The Kalamazoo River bald eagle productivity rate is also one-fifth of the value generally considered to be required to maintain a healthy population, 1.0.

Table 7.13. Comparison of bald eagle nesting success in the Kalamazoo River to other locations

Location	Period of record	Average number reared per active nest
Kalamazoo River	1990-2003	0.2
Fox River/Green Bay	1974-1998	0.7
Coastal Michigan	1974-1997	0.8
Inland Michigan	1974-1997	1.0

Sources: M. Meyer, Wisconsin DNR, personal communication, March 1999; Dave Best, U.S. FWS, personal communication, 2000, 2001, 2002, 2003.

These data indicate that Kalamazoo River eagles are experiencing dramatically decreased reproductive rates compared to bald eagles in reference locations and in other locations known to be contaminated with PCBs. While bald eagle productivity has been shown to be food limited in some areas along the Lake Superior shoreline (Dykstra et al., 1998), similar studies have not found food to be a limiting factor for bald eagles nesting along the coast of Lake Michigan (Dykstra et al., 2001). Furthermore, the dramatic difference in productivity rates between the Kalamazoo River nests and other coastal Lake Michigan and inland Michigan sites indicates that factors other than food availability are most likely limiting bald eagle production in the area. As described below, exposure to PCBs is most likely at least a contributing factor to the decreased reproductive rates of Kalamazoo River bald eagles.

Egg PCB concentrations

PCB concentrations in Kalamazoo River bald eagle eggs are much higher than concentrations in eggs from other Lake Michigan coastal sites or from inland Michigan areas (Table 7.14). Compared to the average PCB concentration in eggs from other Lake Michigan coastal sites, the highest PCB concentration detected in Kalamazoo River eagle eggs, 122 mg/kg (fresh ww), is more than 7 times higher and the average PCB concentration detected in Kalamazoo River eagle eggs, 63 mg/kg (fresh ww) is approximately 4 times higher.

Table 7.14. Concentrations of total PCB and DDE in bald eagle eggs

Location	Year	Total PCB (mg/kg fresh ww)[a]	DDE (mg/kg fresh ww)[a]
Kalamazoo River (Ottawa Marsh)	1994[b]	100/122	8.2/11
	1996[b]	53/32	11/6.1
	2000	32(41)[c]	8.1
	Average	63	8.8
Other Lake Michigan coastal sites, average (range)	1994-2000	17.0 (< 0.0095-34)	4.0 (< 0.0019-8.3)
Inland Michigan lower peninsula sites, average (range)	1994-1997[d]	3.9 (0.84-7.2)	1.1 (0.42-1.75)
Stage I Injury Assessment TRV		20	6.3

a. Values represent calculated estimates of concentrations when freshly laid (Best, 2002).
b. Pairs of eggs collected in 1994 and in 1996 were collected from the same clutch, and thus are not independent samples.
c. Second value is a duplicate analysis of the same egg conducted by Michigan State University Aquatic Toxicology Laboratory (2002e).
d. No eggs were collected from inland Michigan lower peninsula sites from 1998 to 2000.
Sources: Mehne, 2000; Best, 2002; Michigan State University Aquatic Toxicology Laboratory, 2002e.

All five of the Kalamazoo River eagle eggs exceed the hatching success injury threshold of 20 mg/kg (fresh ww). The two eggs collected in 1994 are 5 and 6 times greater than the TRV, and eggs collected in other years are approximately 1.5 to 2.5 times greater. These data indicate that PCB concentrations in the Kalamazoo River bald eagle eggs are sufficient to cause the decreased egg hatching that has been observed in these nests.

Four of the five of the Kalamazoo River bald eagle eggs also have DDE concentrations that exceed the 6.3 mg/kg (fresh ww) TRV from Wiemeyer et al. (1993). However, the PCB egg concentrations are much more elevated relative to the PCB TRV than are the DDE egg concentrations relative to the DDE TRV. Although the available data are not sufficient to determine the relative contributions of PCB and DDE to the observed reductions in Kalamazoo River eagle egg hatchability, the Trustees conclude that PCB concentrations in the eggs are sufficient to cause the observed injury of reduced hatching success in the absence of DDE.

Nestling plasma PCB concentrations

Estimated total PCB concentrations in the two Kalamazoo River bald eagle nestling plasma samples collected from one nest in 1999 are similar to concentrations observed in other Lake Michigan bald eagle nestlings, and higher than those observed in inland locations (Table 7.15). The concentration of PCBs in one nestling plasma sample collected in 2000 was 773.41 µg/kg ww (Michigan State University Aquatic Toxicology Laboratory, 2002e), higher than the

Table 7.15. PCB and DDE concentrations in plasma of nestling bald eagles from the Kalamazoo River and other locations in Michigan and Wisconsin

Location	Year	Number of samples	Sum of 20 PCB congeners (µg/kg ww)	Total PCBs (µg/kg ww)	DDE (µg/kg ww)
Kalamazoo River samples					
Allegan State Game Area[a]	1999	2[b]	80.00 (63.05-96.94)	366.6 (289-444.0)	14.2 (11.9-16.5)
Allegan State Game Area[c]	2000	1	169.89	773.41	—
Mean	1999-2000	2	124.9	570.0	14.2
Geometric mean	1999-2000	2	116.6	532.5	—
Concentrations from selected locations					
Lake Michigan[a]	1999	13[d]	119.3 (11.5-297.4)	546 (53.7-1,360)	56.5 (16.0-128)
Lower Fox River, WI[e]	1991-1995	5	—	267[f] (120-547)	6[f] (< 2.5-54)
Green Bay, WI[e,g]	1987-1995	8	—	207[f] (83-901)	53[f] (4-361)
Lake Erie[h]	1987-1992	35	—	199[f] (81-1,325)	22[f] (< 5-429)
Lake Michigan[h]	1987-1992	25	—	154[f] (14-628)	35[f] (< 5-235)
Lake Superior[h]	1987-1992	45	—	127[f] (12-640)	25[f] (< 5-306)
Lake Huron[h]	1987-1992	12	—	105[f] (5-928)	25[f] (< 5-78)
Inland Wisconsin[i]	1990-1994	38	—	42.6[f] (20.1-262)	3.0[f] (< 2.5-5.5)
Eastern Upper Peninsula, Michigan[h]	1987-1992	16	—	32[f] (< 10-146)	12[f] (< 5-24)
Lower Peninsula, Michigan[h]	1987-1992	49	—	31[f] (< 10-200)	10[f] (< 5-193)
Western Upper Peninsula, Michigan[h]	1987-1992	48	—	25[f] (< 10-177)	10[f] (< 5-245)
Inland Lower Peninsula, Michigan[a]	1999	32	3.8 (ND-31.0)	18.35 (ND-143)	4.5 (ND-18.3)

Table 7.15. PCB and DDE concentrations in plasma of nestling bald eagles from the Kalamazoo River and other locations in Michigan and Wisconsin (cont.)

Location	Year	Number of samples	Sum of 20 PCB congeners (µg/kg ww)	Total PCBs (µg/kg ww)	DDE (µg/kg ww)
Threshold concentrations					
Egg threshold equivalent[j]	—	—	—	189	44
Reduced Great Lakes productivity threshold[k]	—	—	—	125	28

a. Summer et al., 2002. Total PCB concentration was estimated from 20 congener sum. See Section 7.5.1 for explanation.
b. Two 1999 samples from Allegan State Game Area were from same clutch and thus are not independent samples.
c. Michigan State University Aquatic Toxicology Laboratory, 2002e.
d. Does not include two samples from Allegan State Game Area.
e. Dykstra et al., 1996.
f. Geometric mean.
g. Dykstra et al., 2001.
h. Bowerman et al., 2003.
i. Dykstra et al., 1998.
j. Calculated from egg thresholds presented in Section 7.5.2, using relationships developed by Elliott and Harris (2002).
k. Bowerman et al., 2003. Based on relationships between productivity and geometric mean concentrations in plasma of nestling bald eagles within nine subpopulations in the upper Midwest.

concentrations observed at the Lower Fox River and Green Bay, an area also contaminated with PCBs (see Table 7.15). Kalamazoo River nestling plasma PCB concentrations are also higher than the concentration in Great Lakes bald eagle plasma of 125 µg/kg shown by Bowerman et al. (2003) to be correlated with a reproductive rate of 0.7. DDE concentrations in the two plasma samples collected in 1999 were generally lower than those observed in other Lake Michigan bald eagle populations with similar PCB concentrations. They were also lower than the estimated threshold DDE concentration of 28 µg/kg DDE developed by Bowerman et al. (2003). The Kalamazoo River eagle nestling plasma data confirm that nestlings are exposed to PCBs and that their plasma contains concentrations of PCBs that exceed injury thresholds for reduced productivity.

7.6 PCB Concentrations in Eggs of Other Birds

7.6.1 Data sources

The following data sources were used to evaluate reproductive injury to birds other than bald eagles in this section:

- Data on PCB congener concentrations in bird eggs collected by Stratus Consulting in 1995 (A.D. Little, 1996; Midwest Research Institute, 1996)
- Data on PCB concentrations in bird eggs collected by Dr. Charles Mehne (Mehne, 2000)
- Data on PCB concentrations in bird eggs compiled by the MDNR (1987b)
- Data on PCB concentrations in bird eggs collected by Michigan State University Aquatic Toxicology Laboratory (2002d; 2002e).

Stratus Consulting staff sampled bird eggs from five locations in the KRE in 1995 as part of a Trustee study. Samples included one great horned owl egg, five red-winged blackbird eggs, two robin eggs, one wood duck egg, one wood thrush egg, and one yellow warbler egg. These egg samples were analyzed for a suite of 45 PCB congeners (analytical data in A.D. Little, 1996). Split samples from five of these eggs were also analyzed separately for coplanar PCB congeners (PCB 77, 81, 126, and 169) and dioxins/furans (analytical data in Midwest Research Institute, 1996). These results were compiled by Stratus Consulting using the results from Midwest Research Institute for coplanar PCB congeners where available. The sum of the 45 congeners analyzed was assumed to represent the total PCB concentration in these samples.

Additional bird egg PCB and DDE data were obtained from Mehne (2000), the MDNR (1987b), and Michigan State University Aquatic Toxicology Laboratory (2002d, 2002e). Sixteen egg samples from great blue herons, red tailed hawks, great horned owls, and wood ducks were collected from locations in the Allegan State Game Area in 1993 and 1994 and analyzed for total PCBs as Aroclors at the Animal Health Diagnostic Laboratory in Lansing, Michigan (1993 samples) or at the Illinois Department of Agriculture in Centralia, Illinois (1994 samples; Mehne, 2000). Additionally, 14 mute swan eggs were collected in 1986 from the Allegan State Game Area and analyzed for total PCBs (MDNR, 1987b). Two great horned owl eggs were collected in 2002 from the Allegan State Game Area and 3 eastern bluebird eggs, 11 house wren eggs, and 6 tree swallow eggs were collected from Trowbridge in 2001 and analyzed for total PCBs as the sum of congeners (Michigan State University Aquatic Toxicology Laboratory, 2002d, 2002e).

7.6.2 Toxicological benchmarks

Total PCBs

The adverse effects of PCBs in eggs have been determined in both laboratory exposure experiments and field assessments that link measurement endpoints with egg PCB concentrations. Chickens, a particularly sensitive species, experience reduced hatching success, embryo or chick deformities, and other reproductive impairment at concentrations of 1.5 to 5 mg PCB/kg egg ww. In other species of birds, LOELs for the most sensitive endpoint measured range upward from approximately 4 mg PCB/kg egg ww (Table 7.16). NOELs range upward from 1.3 mg PCB/kg egg ww. The data in this table demonstrate the large differences between species' sensitivities to PCBs. For example, the mallard LOEL reported in Table 7.16 exceeds that of the chicken by a factor of more than 50. Many of the study results listed in Table 7.16 may be confounded by the fact that they are based on field studies in which parameters other than PCBs (e.g., other contaminants, hatching and rearing conditions) could not be controlled. Hoffman et al. (1995) selected a TRV range of 8 to 25 mg/kg ww total PCBs in eggs of terns, cormorants, and eagles for the endpoint of reduced hatching success. This TRV range is used for this injury evaluation. Sensitive species may experience reproductive effects at concentrations from 1.5 to 5 mg PCB/kg egg ww.

Table 7.16. Egg total PCB concentrations causing adverse effects

Species	PCB	LOEL mg/kg ww	NOEL mg/kg ww	Adverse effect[a]	Laboratory (L) or field (F) study	Reference
Chicken	Total PCB	1.5	0.95	H	L	Britton and Huston, 1973
Chicken	Total PCB	2.5	0.36	H	L	Scott, 1977
Chicken	Total PCB	4	—	D, H	L	Tumasonis et al., 1973
Bald eagle	Total PCB	4	—	S	F	Ludwig et al., 1993
Caspian tern	Total PCB	4.2	—	S	F	Yamashita, 1993
Chicken	Total PCB	5	< 5	P, F	L	Platonow and Reinhart, 1973
Chicken	A1242	6.7[b]	0.67[b]	G	L	Gould et al., 1997
Chicken	A1254	6.7[b]	0.67[b]	G	L	Gould et al., 1997
Common tern	Total PCB	7	5.2-5.6	H	F	Becker et al., 1993
Bald eagle	Total PCB	7.2	1.3	S	F	Wiemeyer et al., 1984

Table 7.16. Egg total PCB concentrations causing adverse effects (cont.)

Species	PCB	LOEL mg/kg ww	NOEL mg/kg ww	Adverse effect[a]	Laboratory (L) or field (F) study	Reference
Common tern	Total PCB	8	7	S	F	Bosveld and Van den Berg, 1994
Common tern	Total PCB	10	4.8[c]	D, H	L	Hoffman et al., 1993
Bald eagle	Total PCB	13	—	S	F	Wiemeyer et al., 1993
Ringed turtle dove	A1254	16	—	H	L	Peakall and Peakall, 1973
Forster's tern	Total PCB	19	7[c]	S	F	Bosveld and Van den Berg, 1994
Bald eagle	Total PCB	~20	—	S	F	U.S. FWS and Stratus Consulting, 1999
Forster's tern	Total PCB	22.2	4.5	H	F/L	Kubiak et al., 1989
American kestrel	Mixture of Aroclors	34.0	—	P	L	Fernie et al., 2001
Mallard	A1242	105	—	T	F	Haseltine and Prouty, 1980

a. D = embryo or chick deformities; F = reduced fertility; G = reduced chick growth; H = reduced hatching success; P = reduced egg production; S = reduced overall reproductive success; T = egg shell thinning.
b. Concentration is reported as mg/kg, but may actually be in units of µg/kg (K. Grasman, Wright State University, personal communication, 2001).
c. Based on no apparent adverse effects in field population.

TCDD-equivalents

To account for the variations in toxicity of different PCB congeners, potency can be expressed relative to the potency of TCDD, the most toxic, halogenated aromatic compound, using a TEF (see Section 6.3.2 of this document).

As was done for fish, an international group of toxicology experts developed bird TEFs for several PCB and dioxin/furan congeners (Table 7.17). These values were derived from multiple studies and are not specific to any single bird species. The TEFs can be used to calculate TCDD-eq in a sample by multiplying the concentration of each PCB congener by its TEF, and summing the results across all the congeners.

Table 7.17. Avian toxic equivalency factors of PCB, dioxin, and furan congeners relative to TCDD

Compound	Avian TEF
PCB congener 77	0.05
PCB congener 81	0.1
PCB congener 105	0.0001
PCB congener 114	0.0001
PCB congener 118	0.00001
PCB congener 123	0.00001
PCB congener 126	0.1
PCB congener 156	0.0001
PCB congener 157	0.0001
PCB congener 167	0.00001
PCB congener 169	0.001
PCB congener 189	0.00001
2,3,7,8 TCDD	1
2,3,7,8 TCDF	1
1,2,3,7,8 PeCDD	1
1,2,3,7,8 PeCDF	0.1
2,3,4,7,8 PeCDF	1
1,2,3,4,7,8 HxCDD	0.05
1,2,3,6,7,8 HxCDD	0.01
1,2,3,7,8,9 HxCDD	0.1
1,2,3,4,7,8 HxCDF	0.1
1,2,3,6,7,8 HxCDF	0.1
1,2,3,7,8,9 HxCDF	0.1
2,3,4,6,7,8 HxCDF	0.1
1,2,3,4,6,7,8 HpCDD	0
1,2,3,4,6,7,8 HpCDF	0.01
1,2,3,4,7,8,9 HpCDF	0.01
OCDD	0.0001
OCDF	0.0001

Source: Van den Berg et al., 1998.

Numerous studies have been conducted on adverse effects caused by PCB congeners and TCDD in bird eggs. Using TEFs, the PCB congener concentrations reported in these studies have been converted to TCDD-eq in Table 7.18. As the data in Table 7.18 show, chickens are particularly sensitive to TCDD toxicity compared to other bird species studied to date. Egg LD50 concentrations for chickens range from 0.04 to 0.43 µg/kg TCDD-eq, whereas LD50 concentrations for other species range from 1.4 to > 250 µg/kg TCDD-eq (laboratory studies only).

Excluding chicken, reported egg LOEL values from laboratory studies range from 0.23 µg/kg TCDD-eq (for edema in American kestrels) to 4.4 µg/kg TCDD-eq (for embryomortality and deformities in common terns). Therefore, the Trustees select a concentration range of 0.2 to 4 µg/kg TCDD-eq as an estimated range of injury threshold concentrations in bird eggs.

For chickens, the reported egg LOEL values from laboratory studies range from 0.006 µg/kg TCDD-eq to 0.32 µg/kg TCDD-eq. The Trustees select a lower injury threshold range of 0.01 to 0.04 µg/kg TCDD-eq in the egg from the lower end of the range of these LOEL values based on the assumption that differences in LOEL values across the different chicken studies listed in the table are primarily a result of the dosing concentrations and study designs used in the different studies, not because of inherently different sensitivities to TCDD toxicity among the chickens tested. It is the lower end of the reported LOEL range that thus is most relevant for selecting an injury threshold for sensitive species. The value of 0.04 is used as the upper end of the injury threshold range because it corresponds to the lowest reported LD50 value for chicken.

7.6.3 Results

Total PCBs

Measured PCB concentrations in KRE eggs of several avian species were within or above the TRV range for embryomortality (Figure 7.5). Measured concentrations ranged from 8.4 to 14.5 mg/kg ww in eastern bluebird eggs from Trowbridge, 1.5 to 44 mg/kg ww in blue heron eggs from the Allegan State Game Area, from 16 to 91 mg/kg ww in great horned owl eggs, from 2.0 to 8.3 mg/kg ww in house wren eggs from Trowbridge, and from 2.3 to 27 mg/kg ww in red tailed hawk eggs. Eggs from all of the species sampled, except for wood duck and yellow warbler, were within or above the TRV range for sensitive species.

2. No such species have been reported in the literature, although the number of bird species tested to date is limited.

Table 7.18. Bird egg TCDD-eq concentrations causing adverse effects

Species	Toxicant	Measurement	Egg toxicant concentration (µg/kg egg, ww)	TCDD-eq concentration (µg/kg egg, ww)[a]	Adverse effect[b]	Laboratory (L) or field (F) study	Reference
Chicken	TCDD	NOEL	0.1	0.1	H	L	Janz and Bellward, 1996
		NOEL	0.2	0.2	I	L	Peden-Adams et al., 1998
		LOEL[c]	0.006	0.006	D	L	Cheung et al., 1981
		LOEL	0.01	0.01	H	L	Verrett, 1970 (in Hoffman et al., 1996a)
		LOEL	0.04	0.04	H	L	Verrett, 1976 (in Hoffman et al., 1996a)
		LOEL	0.08	0.08	D	L	Walker and Catron, 2000
		LOEL	0.1 (yolk injected)	0.1	G	L	Henshel et al., 1997
		LOEL	0.3 (air cell injected)	0.3	G	L	Henshel et al., 1997
		LOEL	0.32 (yolk injected)	0.32	D	L	Walker et al., 1997
		LD50	0.12 (yolk injected)	0.12	H	L	Henshel et al., 1997
		LD50	0.15	0.15	H	L	Verrett, 1976 (in Hoffman et al., 1996a)
		LD50	0.15	0.15	H	L	Powell et al., 1996
		LD50	0.18 (air cell injected)	0.18	H	L	Henshel, 1993 (in Hoffman et al., 1996a)
		LD50	0.24 (air cell injected)	0.24	H	L	Allred and Strange, 1977 (in Hoffman et al., 1996a)
		LD50	0.3 (air cell injected)	0.3	H	L	Henshel et al., 1997

Table 7.18. Bird egg TCDD-eq concentrations causing adverse effects (cont.)

Species	Toxicant	Measurement	Egg toxicant concentration (µg/kg egg, ww)	TCDD-eq concentration (µg/kg egg, ww)[a]	Adverse effect[b]	Laboratory (L) or field (F) study	Reference
	PCB 77	LD50	2.6	0.13	H	L	Hoffman et al., 1998
		LD50	8.6	0.43	H	L	Brunström and Andersson, 1988
	PCB 105	LD50	2,200	0.22	H	L	Brunström, 1990
	PCB 118	LD50	8,000	0.08	H	L	Brunström, 1989
	PCB 126	LOEL	0.57	0.057	D	L	Walker and Catron, 2000
		LOEL	0.3	0.03	D	L	Hoffman et al., 1998
		LD50	0.4	0.04	H	L	Hoffman et al., 1998
		LD50	1.01	0.101	H	L	Fox and Grasman, 1999
		LD50	2.3	0.23	H	L	Powell et al., 1996
		LD50	3.2	0.32	H	L	Brunström and Andersson, 1988
	PCB 156	LD50	1,500	0.15	H	L	Brunström, 1990
	PCB 157	LD50	2,500	0.25	H	L	Brunström, 1990
	PCB 167	LD50	> 4,000	> 0.04	H	L	Brunström, 1990
	PCB 169	LD50	170	0.17	H	L	Brunström and Andersson, 1988
Osprey	TCDD-eq	NOEL	0.14	0.14	S	F	Woodford et al., 1998
	TCDD-eq	NOEL	~0.05	~ 0.05	H	F	Elliott et al., 2001
Bald eagle	TCDD-eq	NOEL	0.2	0.2	S	F	Elliott et al., 1996
Bobwhite	PCB 126	LD50	24	2.4	H	L	Hoffman et al., 1995

Table 7.18. Bird egg TCDD-eq concentrations causing adverse effects (cont.)

Species	Toxicant	Measurement	Egg toxicant concentration (µg/kg egg, ww)	TCDD-eq concentration (µg/kg egg, ww)[a]	Adverse effect[b]	Laboratory (L) or field (F) study	Reference
Caspian tern	TCDD-eq	NOEL	0.75	0.75	H	F	Ludwig et al., 1993
Domestic pigeon	TCDD	LOEL	3	3	G, H	L	Janz and Bellward, 1996
Eastern bluebird	TCDD	NOEL	1	1	B	F	Thiel et al., 1988
		LOEL	10	10	B	F	
Common tern	PCB 126	LOEL	44	4.4	D, H	L	Hoffman et al., 1998
		LD50	104	10.4	H	L	
	TCDD-eq	NOEL	< 4	< 1 (assuming 25% lipid)	H	L	Bosveld and Van den Berg, 1994
Double-crested cormorant	PCB 126	LD50	158	16	H	L	Powell et al., 1997
		LD50	177	18	H	L	Powell et al., 1998
	TCDD	LOEL	4	4	H	L	Powell et al., 1997
		LD50	4	4	H	L	Powell et al., 1998
	TCDD-eq	LD50	~0.55	0.55	H	F	Tillitt et al., 1992
Forster's tern	TCDD-eq	NOEL	0.05[d]	0.05	H	L/F	Kubiak et al., 1989
		LOEL	0.55[d]	0.55	H	L/F	
Great blue heron	TCDD	NOEL	2	2	H	F	Janz and Bellward, 1996
	TCDD-eq	NOEL	0.02	0.02	G, D	F	Hart et al., 1991
		LOEL	0.245	0.245			
Ring-necked pheasant	TCDD	LOEL	1 (yolk sac injected)	1	H	L	Nosek et al., 1993
		LOEL	1 (albumen injected)	1	H	L	
		LD50	1.4 (albumen injected)	1.4	H	L	
		LD50	2.2 (yolk sac injected)	2.2	H	L	
	PCB 77	NOEL	100	5	H	L	Brunström and Reutergårdh, 1986

Table 7.18. Bird egg TCDD-eq concentrations causing adverse effects (cont.)

Species	Toxicant	Measurement	Egg toxicant concentration (µg/kg egg, ww)	TCDD-eq concentration (µg/kg egg, ww)[a]	Adverse effect[b]	Laboratory (L) or field (F) study	Reference
Wood duck	TCDD-eq	NOEL	≤ 5	≤ 5	H	F	White and Seginak, 1994; White and Hoffman, 1995
		LOEL	> 20-50	> 20-50	H	F	
American kestrel	PCB 77	LD50	316	15.8	H	L	Hoffman et al., 1998
	PCB 126	LOEL	2.3	0.23	D	L	
		LD50	65	6.5	H	L	
Turkey	PCB 77	LD50	~800	40	H	L	Brunström and Lund, 1988
Black-headed gull	PCB 77	LD50	< 1,000	< 50	H	L	Brunström and Lund, 1988
Herring gull	PCB 77	LD50	> 1,000	> 50	H	L	Brunström, 1988
	TCDD-eq	NOEL	1-2	1-2	H	F	Ludwig et al., 1993
Domestic goose	PCB 77	LD50	> 1,000	> 50	H	L	Brunström, 1988
Goldeneye	PCB 77	LD50	> 1,000	> 50	H	L	Brunström and Reutergårdh, 1986
Mallard	PCB 77	LD50	> 5,000	> 250	H	L	Brunström, 1988

a. Calculated using TEFs (Van den Berg et al., 1998).

b. A = adult mortality; B = reproductive behavior; D = deformities; F = female fertility; G = chick growth; H = hatching success; I = immunological changes; M = male fertility; P = egg production; S = population size or reproductive success; T = egg shell thinning. Data are organized by the general rank order of TCDD-eq toxicity values.

c. This LOEL is an effective dose associated with a 20% increase in heart deformities (ED-20) derived from a statistically significant log dose response regression.

d. TCDD-eqs from Kubiak et al., 1989, were originally reported using TEFs from Sawyer and Safe (1982) based on testing in rats as NOEL = 0.2 and LOEL = 2.2. TCDD-eqs were recalculated using the WHO avian TEFs (Table 7.17).

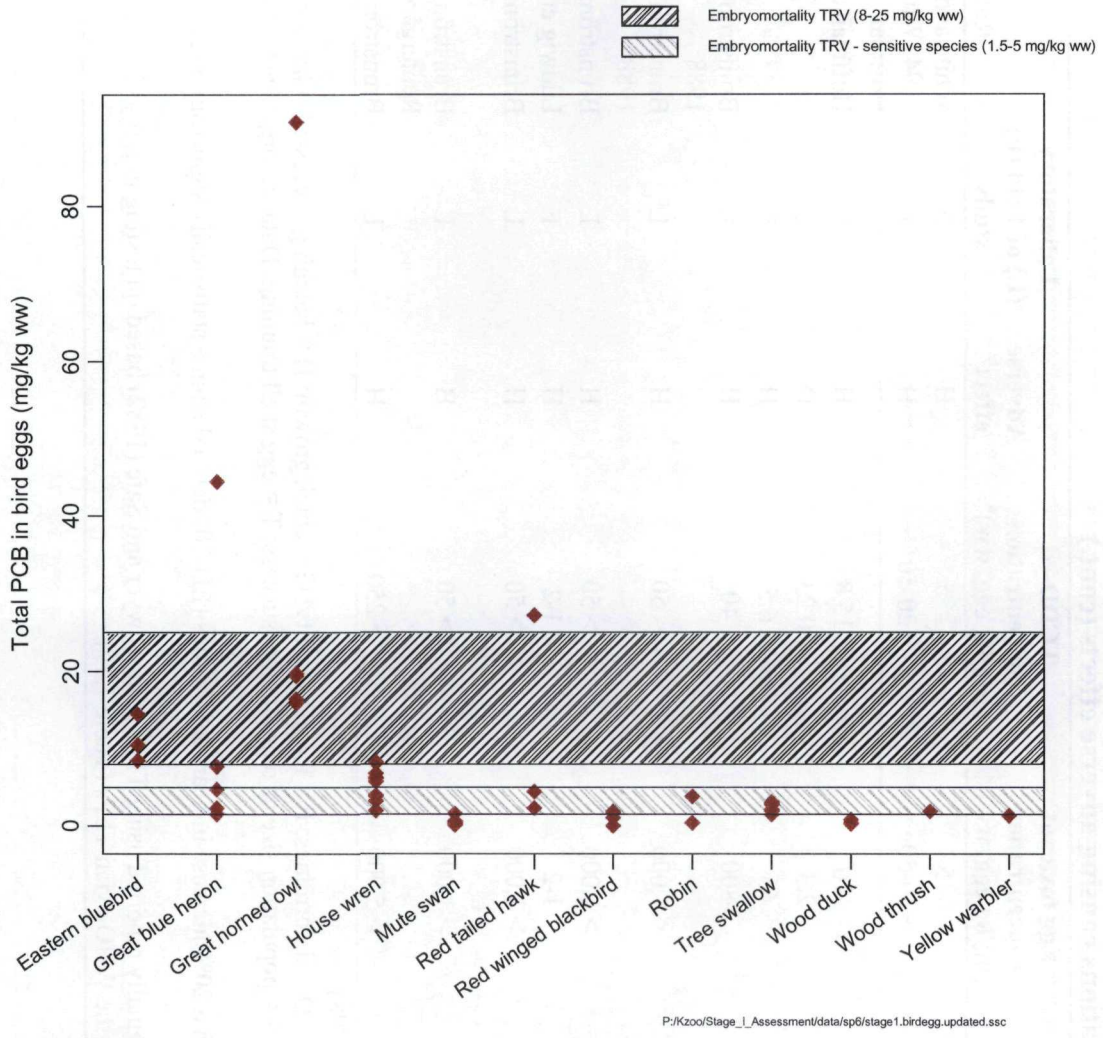

Figure 7.5. Total PCB concentrations in bird eggs collected in Allegan State Game Area and other locations along the Kalamazoo River.

Sources: Michigan Department of Natural Resources, 1987b; A.D. Little, 1996; Midwest Research Institute, 1996; Mehne, 2000; Michigan State University Aquatic Toxicology Laboratory, 2002c.

TCDD-equivalents

Measured TCDD-eq concentrations from PCBs in KRE bird eggs were above the range of embryomortality for sensitive species in 5 of 11 samples (3 red-winged blackbirds, 1 wood duck, and 1 great horned owl), and within the range in 1 red-winged blackbird sample (Figure 7.6). The maximum TCDD-eq from PCBs was 0.175 µg/kg in 1 great horned owl egg collected near Lake Allegan Dam. TCDD-eqs in other red-winged blackbird, robin, wood thrush, and yellow warbler eggs were less than 0.0074 µg/kg. TCDD-eqs from dioxins and furans were analyzed for four of the eggs sampled. PCBs accounted for 66 to 95% of the total TCDD-eq in these eggs because of the much higher concentrations of PCBs than dioxins or furans in these samples.

These results indicate that at least some avian species in addition to bald eagles (discussed in Section 7.5 of this document) may be injured by PCBs. Concentrations of total PCBs and TCDD-eqs in the eggs of several species are within the range associated with embryomortality in sensitive species. Some great blue heron, great horned owl, and red tailed hawk eggs also have PCB concentrations within the range associated with embryomortality in less sensitive species. However, the limited nature of these data make it difficult to identify the temporal and geographical extent of injury.

7.7 Total PCBs in Mammalian Diets

In this section, the results of the MDEQ ERA for mammals exposed to PCBs in their diet are discussed, and PCB concentrations in KRE fish are compared to TRVs for piscivorous mammals such as mink.

7.7.1 Data sources

Some of the same data sources used to evaluate injury to birds in Section 7.4 are also used to evaluate injury to mink in this section. As for avian species (see Section 7.4.1), the MDEQ calculated estimated exposure concentrations in the diets of mammalian species of concern based on dietary exposure models (Camp Dresser & McKee, 2003b). Mammalian species evaluated in the ERA were mink, white footed/deer mouse, muskrat, and red fox. Using dietary no effect and low effect values derived from the literature, MDEQ then calculated no effect – and low effect hazard quotients to characterize risk from PCB exposure for these species. PCB concentrations measured in KRE whole-body fish are compared to the dietary TRVs for mink. These data are described in Section 7.4.1.

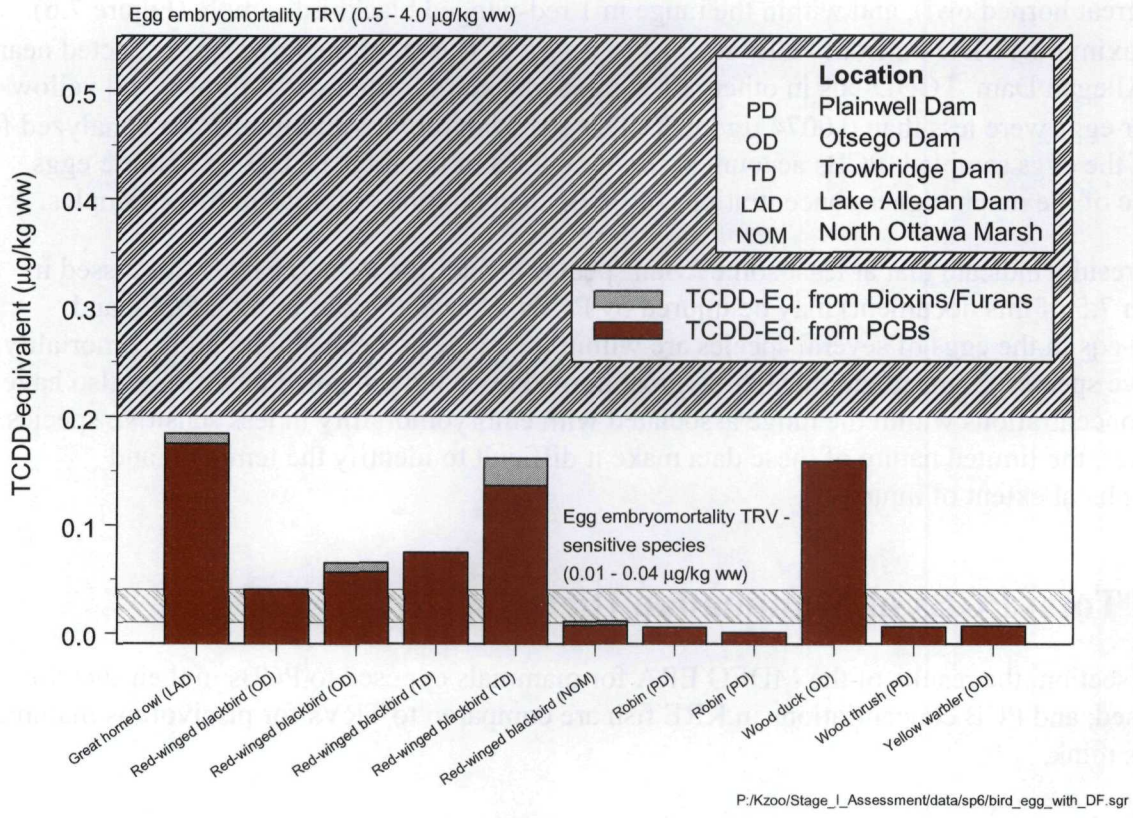

Figure 7.6. TCDD-eq concentrations based on PCBs and dioxins/furans measured in individual bird eggs in the KRE compared to TRVs for egg embryomortality. Dioxins and Furans were only measured in four samples (one great horned owl and three red winged blackbird). Contributions to toxicity by dioxins and furans in the remaining samples is unknown.

Sources: A.D. Little, 1996; Midwest Research Institute, 1996.

7.7.2 Toxicological benchmarks

Stage I Injury Assessment TRVs were developed for mink only, since mink are expected to be highly exposed to PCBs through their diet of fish and other aquatic biota and they are sensitive to PCB toxicity. The mink TRVs were developed from controlled laboratory dietary toxicity tests reported in the literature where PCBs were administered in mink diets. In many of these studies, measurement endpoints included sublethal effects such as increased organ weights, altered enzyme activity, and depressed growth. Reproductive effects such as reduced or eliminated reproduction, reduced kit weight and growth, and kit mortality were also monitored.

In adult mink, female reproductive endpoints are generally the most sensitive endpoints to PCB toxicity (Peterson et al., 1993). Dietary PCB concentrations as low as 0.25 mg/kg diet ww have been found to cause delayed estrus and a reduced whelping rate in mink fed PCB contaminated carp from the Great Lakes (Restum et al., 1998). Hornshaw et al. (1983) and Platonow and Karstad (1973) found more severe reproductive effects such as reduced reproduction and kit mortality at LOELs of 0.66 and 0.64 mg/kg diet ww, respectively. The Hornshaw et al. (1983) study fed adult females PCB contaminated fish from the Great Lakes, while the Platonow and Karstad (1973) study fed mink beef from cattle that had been fed a diet laden with PCBs. Similar effects on kit survival were seen by Heaton et al. (1995a) at a LOEL of 0.72 mg/kg diet ww, and by Wren et al. (1987a, 1987b), Aulerich and Ringer (1977), and Restum et al. (1998) at a LOEL of 1.0 mg/kg diet ww. Complete elimination of reproduction is associated with dietary PCB concentrations of 1.5 mg/kg diet ww (Hornshaw et al., 1983). The low effect value for mink used in the Ecological Risk Assessment (Camp Dresser & McKee, 2003b) was 1.1 mg/kg ww. For this Stage I Assessment, a mink dietary TRV range of 0.5 to 1.0 mg/kg ww total PCBs in diet is used to evaluate injury to mink through dietary exposure. Concentrations in diet within this range would be expected to cause reduced reproduction or kit mortality in mink. Although TCDD-eq based dietary TRVs for mink are also available (e.g., Brunström et al., 2001), almost all of the available KRE PCB concentration data for potential mink dietary items, such as fish, are quantified as total PCBs or Aroclor mixtures, making TCDD-eq based TRVs difficult to use.

7.7.3 Results

The MDEQ (Camp Dresser & McKee, 2003b) calculated risks to mammals through dietary exposure by dividing the average estimated daily exposure concentration by NOEL and LOEL values (Table 7.19). The ERA concluded that risks to sensitive piscivorous predators, such as mink, are high. MDEQ concluded that carnivorous mammals such as red fox are not likely to be at significant risk unless foraging is concentrated in riparian areas with contaminated floodplain sediment and the fox diet consists of prey that have taken up substantial amounts of PCBs. Omnivorous species such as mice are not likely to be at risk through dietary exposure pathways

Table 7.19. Summary of risks to terrestrial wildlife from the ERA (MDEQ, 2003)

Species	NOEL-based hazard quotient[a]	LOEL-based hazard quotient[a]
Mink	19	15
White footed/deer mouse	0.7	0.2
Muskrat	0.3	0.08
Red fox	2.5	0.5

a. Risks are summarized as hazard quotients, which are calculated by dividing a NOEL or LOEL by the estimated daily exposure concentration. The mink NOEL is the exposure concentration predicted to cause a 10% decrease in reproduction, and the mink LOEL a 25% decrease (Camp Dresser & McKee, 2003b). For the other species, the effect endpoints vary. NOEL or LOEL values greater than 1 mean that dietary PCB exposure exceeds the TRV.

Source: Camp Dresser & McKee, 2003b.

unless they reside in the most contaminated areas. The hazard quotients for muskrat, a semi-aquatic herbivorous mammal, are also low. However the TRVs for muskrat were based on threshold values for rats which may not be equally sensitive to PCBs via ingestion. Overall, the MDEQ ERA concludes that the KRE has been, and is currently being adversely affected by PCBs.

A comparison of whole fish PCB concentrations with TRVs for mink dietary exposure is consistent with the conclusions of the ERA (Figure 7.7). Many of the samples from all years are within or above the range expected to cause reproductive toxicity to mink. Overall, 199 of the 228 fish samples (87%) collected are within or above the mink TRV range. Fish containing PCBs sufficient to cause reproductive effects in mink are distributed throughout the KRE, including Portage Creek and the Kalamazoo River from the city of Kalamazoo to Lake Michigan. These conditions have been observed since as early as 1993 and have persisted through 2000. However, it is likely that fish have accumulated PCBs since the time of the initial releases, and also likely that concentrations were much higher in the past than those measured in 1993.

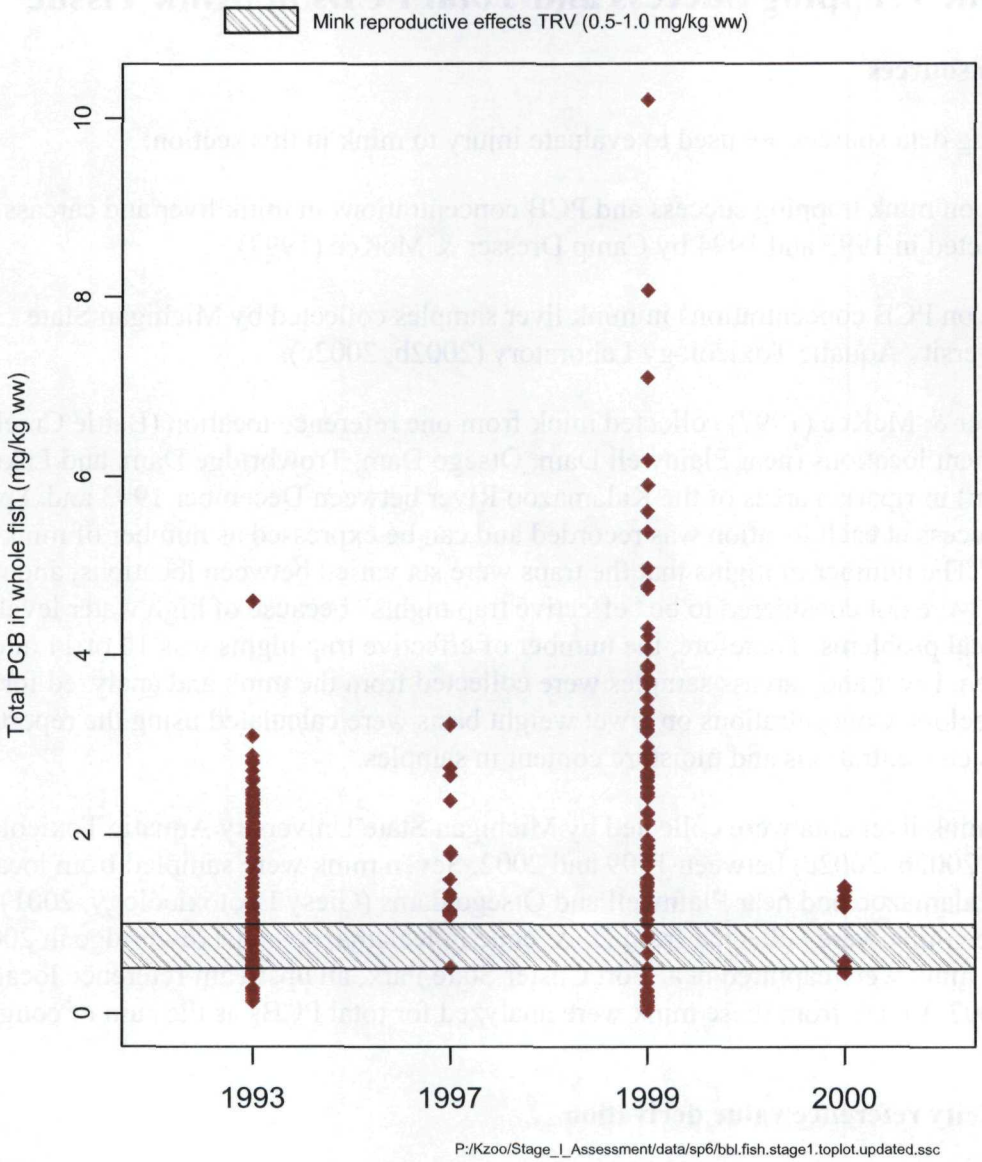

Figure 7.7. Total PCBs in whole body fish samples collected in the Kalamazoo River downstream of PRPs compared to TRVs for reproductive effects in mink. Species include channel catfish, golden redhorse, northern hogsucker, smallmouth bass, spotted sucker, and white sucker.

Sources: Blasland, Bouck & Lee 2001; Camp Dresser & McKee, 2002b; Michigan State University Aquatic Toxicology Laboratory, 2002j.

7.8 Mink Trapping Success and Total PCBs in Mink Tissue

7.8.1 Data sources

The following data sources are used to evaluate injury to mink in this section:

- Data on mink trapping success and PCB concentrations in mink liver and carcass samples collected in 1993 and 1994 by Camp Dresser & McKee (1997)
- Data on PCB concentrations in mink liver samples collected by Michigan State University Aquatic Toxicology Laboratory (2002b, 2002c).

Camp Dresser & McKee (1997) collected mink from one reference location (Battle Creek) and four assessment locations (near Plainwell Dam, Otsego Dam, Trowbridge Dam, and Lake Allegan Dam) in riparian areas of the Kalamazoo River between December 1993 and April 1994. Trapping success at each location was recorded and can be expressed as number of mink per "trap night." The number of nights that the traps were set varied between locations, and many of these nights were not considered to be "effective trap nights" because of high water levels or other logistical problems. Therefore, the number of effective trap nights was 10 or 11 at each sampling area. Liver and carcass samples were collected from the mink and analyzed for total PCBs as Aroclors. Concentrations on a wet weight basis were calculated using the reported dry weight basis concentrations and moisture content in samples.

Additional mink liver data were collected by Michigan State University Aquatic Toxicology Laboratory (2002b, 2002c) between 1999 and 2002. Seven mink were sampled from locations in the city of Kalamazoo and near Plainwell and Otsego dams (Giesy Ecotoxicology, 2001). Another three mink were collected from D. Avenue in Kalamazoo and Trowbridge in 2001 and 2002. Three mink were captured near Fort Custer State Park, an upstream reference location in 2001 and 2002. Livers from these mink were analyzed for total PCBs as the sum of congeners.

7.8.2 Toxicity reference value derivation

Few studies have evaluated toxic effects associated with PCB concentrations in mink carcasses. Leonards et al. (1995) calculated whole body concentrations of total PCBs in mink from experimental literature data. Whole body EC_{50} concentrations (concentrations at which 50% of the subjects experience the tested effect) for reduced litter size and kit survival were estimated from these studies. The calculated EC_{50} concentration for reduced litter size was 1.2 µg/g PCB ww, in mink whole body samples and concentrations greater than 20 µg/g were associated with complete reproductive failure. The mean lipid weight of muscle was 2 to 3%, which translates the EC_{50} to 40 to 60 µg/g on a lipid normalized basis, similar to the EC_{50} of 65 µg/g (lipid) for

reduced litter size reported by Kihlström et al. (1992). Leonards et al. (1995) also calculated an EC_{50} of 2.36 µg/g ww for kit survival based on predicted whole body concentrations from the results of experimental literature. The EC_{50} whole body PCB concentration of 1.2 µg/g ww for reduced litter size and the EC_{50} of 2.36 µg/g ww for kit survival are used to evaluate injury in whole body mink in this Stage I Assessment, although it should be recognized that the injuries at these concentrations are severe (50% reduction in litter size or kit survival), and thus these values are not equivalent to LOEL values.

Tissue PCB concentrations associated with adverse effects are also available for mink livers (Kannan et al., 2000). Adverse reproductive effects have been observed at adult mink liver PCB concentrations of 0.2 to 7.25 mg/kg ww (Platonow and Karstad, 1973; Den Boer, 1984; Wren et al., 1987a, 1987b; Heaton et al., 1995a; Restum et al., 1998; Halbrook et al., 1999). With the exception of one study (Halbrook et al., 1999), all the LOEL concentrations in mink livers for reproductive effects are less than 2.2 mg/kg ww. Halbrook et al. (1999) evaluated the effects of a diet of PCB contaminated fish from near Oak Ridge, Tennessee, on ranch mink, and found a LOEL for reproductive effects of 7.25 mg/kg PCBs ww in adult livers. This higher LOEL value may be the result of a different PCB congener profile or study design. A reproductive effects TRV range of 0.2 to 2.2 mg/kg ww for reproductive effects is used to evaluate injury in mink livers in this Stage I Assessment. Studies on mortality effects associated with PCBs in mink livers report LOEL concentrations from 4.2 to 12.0 mg/kg ww (Aulerich et al., 1973; Platonow and Karstad, 1973; Kubiak and Best, 1991). This range is also used to evaluate injury to mink in this Stage I Assessment.

7.8.3 Results

Trapping success

Mink trapping success was lower in assessment areas than in the upstream reference location in Battle Creek (Table 7.20). Under a similar trapping effort, five mink were caught at Battle Creek whereas no mink were captured near Otsego Dam, one was captured upstream of Plainwell Dam, and two were captured at both Trowbridge Dam and Lake Allegan Dam. Camp Dresser & McKee (1997) attributed the reduced trapping success to reduced mink populations in the sampling locations within the assessment areas. There was suitable mink habitat throughout the study area, apparently adequate food supply, and equal trapping effort among sites. This study was not designed to sample mink populations, rather to collect mink for tissue sampling; however, it provides supporting evidence that KRE mink populations are adversely affected by PCB contamination in the Kalamazoo River.

Table 7.20. Mink trapping success, 1993-1994

Sampling location	Number of mink caught	Number of effective trap nights
Battle Creek (reference)	5	10
Upstream of Plainwell Dam	1	10
Downstream of Otsego Dam	0	10
Upstream of Trowbridge Dam	2	11
Downstream of Lake Allegan Dam	2	11

Source: Camp Dresser & McKee, 1997.

Total PCBs

PCB concentrations in mink tissue indicate that injury may be occurring; however, few samples have been analyzed. The concentrations of PCBs in all five carcasses from assessment areas are higher than the concentration associated with 50% reduction in kit survival, and four of five are higher than the concentration associated with 50% reduction in litter size (Figure 7.8). In contrast, three of the four samples from the reference location are below the concentration associated with reduced kit survival and all are lower than the concentration associated with reduced litter size.

Livers from the same mink collected in 1994 and others sampled between 1999 and 2002 show similar results (Figure 7.9). All of the livers from mink sampled in 1994 have concentrations above the range associated with reproductive effects, and the concentration of PCBs in one sample, at 12.5 mg/kg ww, is higher than the mortality effects TRV. Livers from mink collected from the KRE assessment area between 1999 and 2002 range in PCB concentration from 0.03 to 6.03 mg/kg ww, with eight out of the ten samples falling within the TRV range. The samples collected in the Fort Custer reference location have concentrations from 1.55 to 3.68 mg/kg ww.

These tissue concentration results suggest that KRE mink are injured by exposure to PCBs. This conclusion is consistent with the analysis of PCBs in KRE mink diet (Section 7.7) and the observed reduced mink trapping success. However, the tissue data are insufficient to draw any conclusions about the geographic or temporal extent of injury.

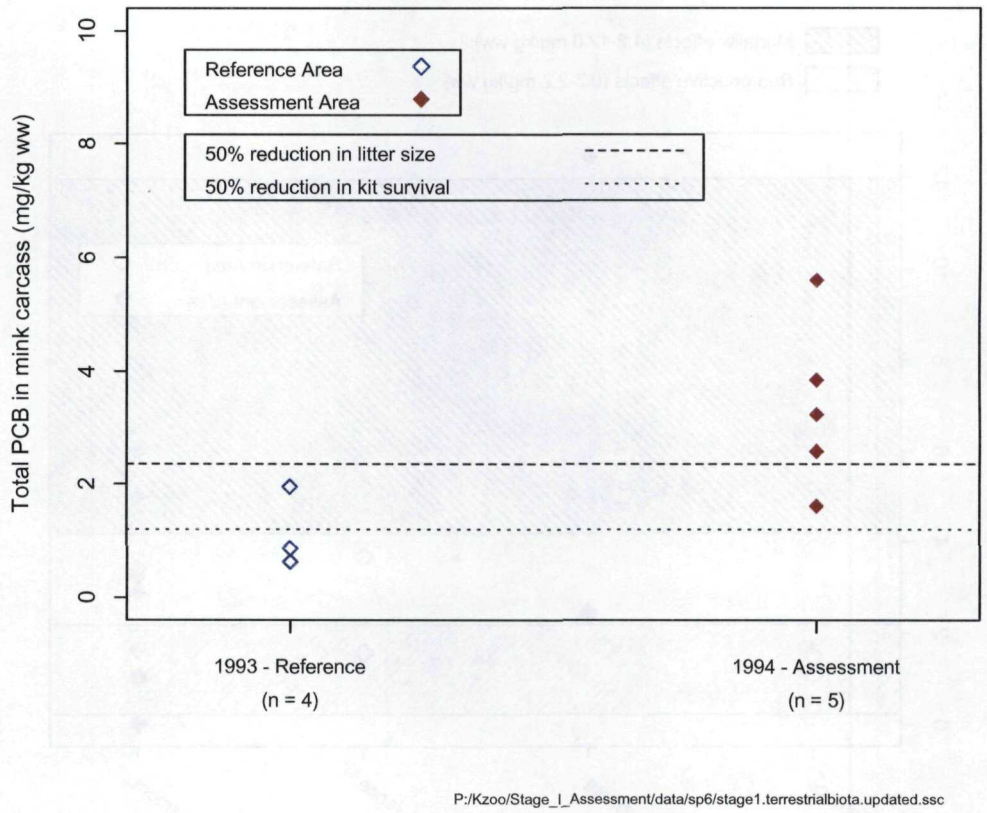

Figure 7.8. Total PCB concentrations in mink carcasses collected in reference and assessment locations. Two of the four reference samples collected in 1993 have concentrations of approximately 0.63 mg/kg ww and overlap in this figure.

Source: Camp Dresser & McKee, 1997.

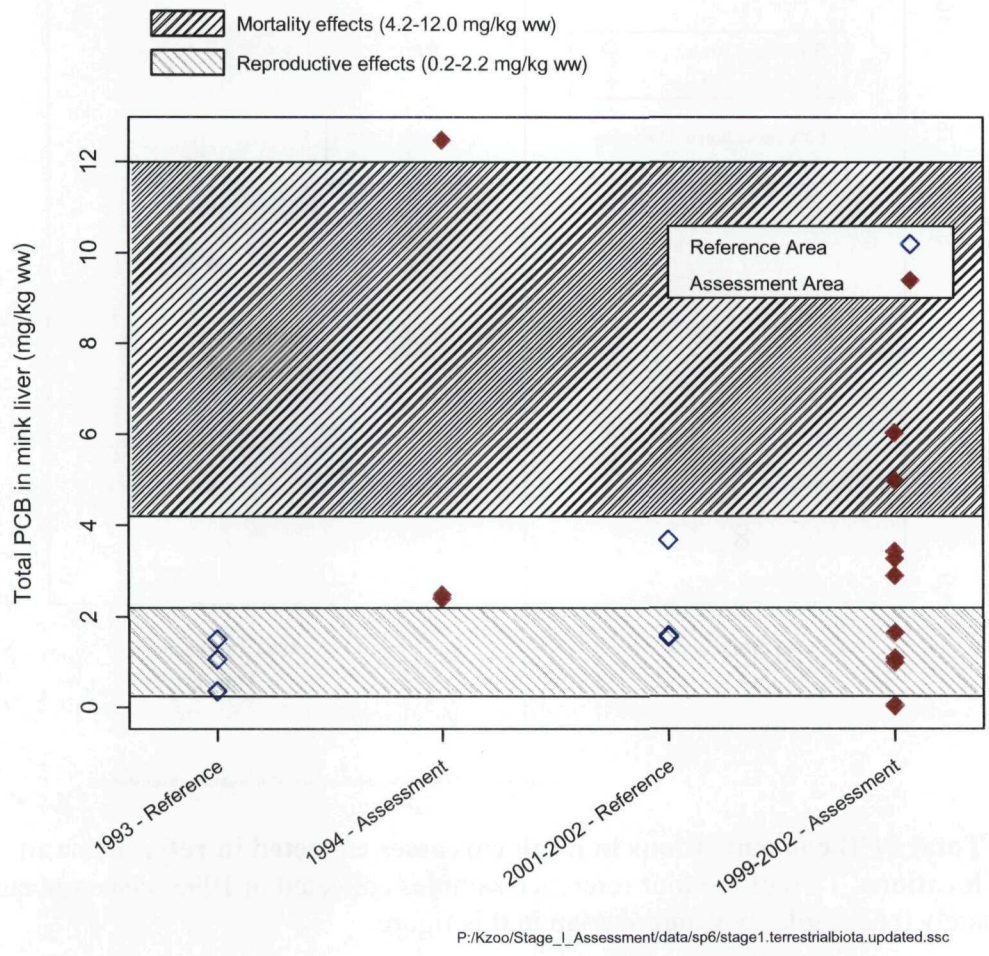

Figure 7.9. PCB concentrations in mink livers from reference and assessment locations compared to injury threshold ranges.

Sources: Camp Dresser & McKee, 1997; Michigan State University Aquatic Toxicology Laboratory, 2002b.

7.9 Total PCBs in Small Mammal, Shrew, and Muskrat Tissue

7.9.1 Data sources

The following data sources are used to evaluate injury to mice and muskrat in this section:

- PCB concentrations in mice collected in 1993 by Blasland, Bouck & Lee (2001)
- PCB concentrations in small mammals and shrews collected in 2000 by Michigan State University Aquatic Toxicology Laboratory (2002f; 2002g; 2002h; 2002i)
- PCB concentrations in muskrat livers collected in 1993 and 1994 by Camp Dresser & McKee (1997)
- PCB concentrations in whole body muskrat samples collected from 2000-2002 by Michigan State University Aquatic Toxicology Laboratory (2002c).

Blasland, Bouck & Lee (1994d) collected deer mice at one reference and four assessment locations along the Kalamazoo River in 1993. The reference location was upstream of Battle Creek. The assessment locations were near Plainwell Dam, Otsego Dam, Trowbridge Dam, and Lake Allegan Dam. Traps were set at 10 meter intervals across a grid at each of the sampling areas. The initial grids contained 48 traps each, and were extended spatially, with additional traps, until 10 mice were collected at each location. Whole body samples were analyzed (without gut contents) for PCB concentrations as Aroclors.

Michigan State University Aquatic Toxicology Laboratory (2002f, 2002g, 2002h, 2002i) collected a variety of small mammal species from a reference location in Fort Custer and an assessment location near Trowbridge Dam in 2000. A total of 34 deer mouse, eastern chipmunk, jumping mouse, red squirrel, and shrew (order Insectivora) samples were collected from Fort Custer. A total of 38 deer mouse, meadow vole and shrew samples were collected from near Trowbridge Dam.

In conjunction with the mink trapping study discussed in Section 7.8.1, Camp Dresser & McKee (1997) collected muskrat from one reference location (Battle Creek) and four assessment locations (near Plainwell Dam, Otsego Dam, Trowbridge Dam, and Lake Allegan Dam). Traps were set in riparian areas of the Kalamazoo River between December 1993 and April 1994. Six muskrat were collected at each sample location. Liver tissue samples were analyzed for total PCBs as Aroclors. One liver sample from Trowbridge Dam was lost; thus there are only five liver samples from this location.

Whole body muskrat samples were collected by Michigan State University Aquatic Toxicology Laboratory (2002c) from a reference location in Fort Custer and an assessment location in Trowbridge. Three samples were collected in Fort Custer in 2000, and one in 2002. Four samples

were collected at Trowbridge Dam in 2000 and three in 2001. These samples were analyzed for total PCBs as the sum of congeners.

7.9.2 Toxicity reference value derivation

Many studies have been conducted on the toxicity of PCBs to mammals. Since mink are the most sensitive mammal tested to date, the TRV concentrations used for mink (see Section 7.8.2) are used to evaluate injury to mice and muskrat as well. If concentrations in deer mouse and muskrat tissue are higher than those expected to cause reproductive effects in mink, further evaluation will be warranted. If they are lower than the mink TRVs, it would be reasonable to conclude that there is no evidence of injury to these species. Thus, whole body TRVs of 1.2 mg/kg ww (reduced reproduction) and 2.36 mg/kg ww (survival of offspring) are compared to whole body PCB concentrations in mice and muskrats, and a reproductive effects TRV range of 0.2 to 2.2 mg/kg ww in liver is compared to muskrat liver PCB concentrations.

7.9.3 Results

PCB concentrations measured in deer mice collected in 1993 and in deer mice, eastern chipmunk, meadow voles, and shrews collected in 2000 are slightly higher in assessment areas than in the reference areas (Table 7.21). However, PCB concentrations in only seven shrew samples of the 77 total small mammal and shrew samples from locations downstream of PRPs are near those associated with reproductive effects in mink. Three of these samples exceed the TRV associated with reduced survival of mink offspring (2.36 mg/kg ww). The maximum concentration in a whole body shrew sample is 3.15 mg/kg ww. However, the sensitivity of shrews to PCB toxicity is unknown. The available data do not indicate that most small mammals are injured by exposure to PCBs, although injury to shrews (Insectivora) is somewhat uncertain.

Measured PCB concentrations are higher in muskrat livers in assessment areas than in the reference location (Table 7.22). PCBs were not detected in the six muskrat livers collected from the Battle Creek reference location, and concentrations range from 0.03 to 1.18 mg/kg ww in assessment area samples. A muskrat liver sample from near Trowbridge Dam has the highest concentration, 1.18 mg/kg ww. Mean concentrations in assessment area muskrat livers are within the range of concentrations expected to be associated with toxic effects in mink. No PCB toxicity tests on muskrat are available to assess muskrat sensitivity to PCBs, but the muskrat is a rodent, and rodents tend to be less sensitive to PCB toxicity than mink (Shore and Douben, 1994). Whole body muskrat samples, however, do not exceed whole body mink reproductive effects TRVs (Table 7.23). Therefore, the available data do not conclusively indicate that the PCB concentrations measured in muskrat tissue are sufficient to cause injury to the muskrat, and this is therefore an area of uncertainty.

Table 7.21. Total PCB concentrations in small mammals and shrews

Location	Species	Number of samples	Total PCB concentration in whole body (mg/kg ww)[a]	Maximum exceeds mink reproductive effects TRV?[b]	Sample Year[c]
Battle Creek (reference)	Deer mouse	10	0.01 (0.01-0.04)	No	1993
Fort Custer (reference)	Deer mouse	9	0.01 (0.00-0.03)	No	2000
Fort Custer (reference)	Eastern chipmunk	5	0.00 (0.00-0.01)	No	2000
Fort Custer (reference)	Jumping mouse	3	0.08 (0.01-0.18)	No	2000
Fort Custer (reference)	Red squirrel	1	0.00	No	2000
Fort Custer (reference)	Shrew	16	0.01 (0.00-0.02)	No	2000
Upstream of Plainwell Dam	Deer mouse	10	0.09 (0.01-0.28)	No	1993
Downstream of Otsego Dam	Deer mouse	10	0.26 (0.09-0.38)	No	1993
Upstream of Trowbridge Dam	Deer mouse	10	0.12 (0.01-0.45)	No	1993
Trowbridge	Deer mouse	11	0.17 (0.02-0.55)	No	2000
Trowbridge	Eastern Chipmunk	1	0.57	No	2000
Trowbridge	Meadow vole	9	0.04 (0.01-0.08)	No	2000
Trowbridge	Shrew	17	1.31 (0.03-3.15)	Yes	2000
Downstream of Lake Allegan Dam	Deer mouse	10	0.06 (0.01-0.35)	No	1993

a. Mean (range). One-half the detection limit was used to calculate the mean if a concentration was below the analytical detection limit.
b. Whole body mink TRVs are 1.2 mg/kg PCBs for reduced litter size and 2.36 for reduced kit survival (Section 7.8.2).
c. Samples collected in 1993 from Blasland, Bouck & Lee (2001). Samples collected in 2000 from Michigan State University Aquatic Toxicology Laboratory (2002f, 2002g, 2002h, 2002i).

Table 7.22. Total PCB concentrations in muskrat livers

Location	Number of samples	Total PCB in liver (mg/kg ww)[a]	Maximum falls within mink reproductive effects TRV range (0.2-2.2 mg/kg)?
Battle Creek (reference)	6	ND	No
Upstream of Plainwell Dam	6	0.30 (0.03-0.70)	Yes
Downstream of Otsego Dam	6	0.13 (0.03-0.27)	Yes
Upstream of Trowbridge Dam	5	0.44 (0.06-1.18)	Yes
Downstream of Lake Allegan Dam	6	0.33 (0.09-0.49)	Yes

a. Mean (range). ND = not detected at detection limit of 0.02 or 0.03 mg/kg ww. Concentrations on a wet weight basis were calculated using the reported dry weight basis concentrations and moisture content in samples.

Source: Camp Dresser & McKee, 1997.

Table 7.23. Total PCB concentrations in whole body muskrat samples collected between 2000 and 2002

Location	Number of samples	Total PCB (mg/kg ww)[a]	Maximum exceeds mink reproductive effects TRV (1.2 mg/kg)?
Fort Custer (reference)	4	0.01 (0.01-0.03)	No
Trowbridge Dam	7	0.07 (0.01-0.11)	No

a. Mean (range).

Source: Michigan State University Aquatic Toxicology Laboratory, 2002c.

7.10 Bioaccumulation of PCBs from Floodplain Soils

The assessment of injuries to KRE wildlife in the preceding sections is based primarily on PCB concentrations measured in biota tissue samples (fish, bird eggs, and mammal tissue). However, these data samples represent only a small fraction of the organisms exposed to PCBs in the KRE, and in only a few selected areas. To address the uncertainty associated with the limited nature of the available tissue data, this section presents an evaluation of potential wildlife injuries based on the much more extensive KRE floodplain soil PCB concentration data that are available. A bioaccumulation exposure model is used to estimate soil concentrations at which food chain exposure for selected wildlife species would cause adverse toxicological impacts, and the estimated soil effects PCB concentrations are compared to the available data on PCB concentrations in KRE soils.

This analysis not only expands the scope of the Stage I KRE wildlife injury assessment but it also can serve as the foundation for restoration scaling approaches that rely on estimates of spatial extent and degree of injury, such as Habitat Equivalency Analysis (HEA; NOAA, 1997). HEA is a tool that can be used to determine the type and extent of habitat restoration that is necessary to offset injuries to natural resources caused by hazardous substances. HEA balances the duration, degree, and spatial extent of habitat services lost because of the injuries with the duration, degree, and spatial extent of habitat services gained through habitat restoration. The spatially-based analysis presented in this section can be used to help define the floodplain habitat services lost because of the presence of PCBs in the soils and the potential for the PCBs to cause adverse effects on wildlife.

7.10.1 Data sources

The following data sources are used in this evaluation:

- Floodplain surface soil samples collected at depths of 0-6 in. by Blasland, Bouck & Lee (2001) in July and August 1993, and analyzed for PCBs as Aroclors. Methods are detailed in Blasland, Bouck & Lee (1994f).

- Floodplain surface soil samples collected at depths of 0-6 in. by Blasland, Bouck & Lee (2001) between November 1993 and February 1994, and analyzed for PCBs as Aroclors. Methods are detailed in Blasland, Bouck & Lee (1994c).

- Floodplain surface soil samples collected as part of a focused sampling program designed by MDEQ (Blasland, Bouck & Lee, 2000b, 2001). Samples were collected from April to August 2000 and analyzed for PCBs as Aroclors. The sample locations were selected by MDEQ to characterize known and suspected PCB point sources and wildlife habitat areas.

- Floodplain surface soil samples from the Plainwell and Otsego City impoundments collected at depths of 0-6 in. in the spring and summer of 2001 as part of the Stage I Removal Assessment by R.F. Weston for EPA and Camp Dresser & McKee (2002a) for MDEQ. Sampling was designed to supplement information provided by existing samples (U.S. EPA and MDEQ, 2002). Initial samples were collected on a grid across the former impoundments, and then additional samples were collected in clusters to better define the PCB distribution observed in grid samples.

Because terrestrial biota generally are exposed to surface soils, only surface samples from these studies were considered. The majority (91%) of these surface samples were collected at depths of 0-6 inches. Surface samples for which an entire 0-6 inch section was not available, or for which a deeper surface section (up to 14 inches) was collected, were also used in this analysis. Although

soils at depths greater than 6 inches may become available to biota via burrowing or human disturbance, it is assumed that PCBs in this surface layer are the best estimate of what is biologically available.

Only soil samples collected from the Kalamazoo River floodplain or from one of the former impoundments are included in this analysis. Former impoundment and river boundaries were defined using historical and current aerial photographs (USGS, 1956, 1965; Air-Land Surveys, 1999). Samples were identified as being within one of the three former impoundments if they were located within these defined boundaries and were not located within the river channel. Samples were identified as being within the Otsego City impoundment if they were located upstream of the Otsego Dam and downstream of the former Plainwell Dam. Samples were identified as being within the Kalamazoo River floodplain if they were not located within one of the former impoundments or the Otsego City impoundment, and if they were identified as Kalamazoo River floodplain samples by Blasland, Bouck & Lee (2001). No other available sample data were included in the analysis of floodplain soil data.

7.10.2 Toxicity reference value derivation

There are no state or federal regulatory standards for the protection of biological resources from hazardous substance concentrations in soil. However, non-enforceable guidelines for PCB concentrations in soil that are protective of ecological receptors are available from several sources.

The DOI (as cited in U.S. EPA, 1990) has found that, generally, PCB concentrations in soils of less than 1-2 mg/kg dw are protective of wildlife. Additionally, preliminary remediation goals (PRGs) have been compiled by the U.S. Department of Energy (DOE) (Efroymson, et al., 1997). These PRGs are concentrations that are anticipated to protect ecological endpoints and were extracted largely from toxicological benchmarks developed for the Oak Ridge National Laboratory (ORNL). The PRG for PCBs in soil, 0.371 mg/kg dw, is not intended to be protective of a specific species, but is the lowest value of PRGs developed for wildlife, plants, and soil invertebrates.

The food web model that was developed for the KRE by the MDEQ in its ecological risk assessment (Camp Dresser & McKee, 2003b) was used to calculate soil concentrations that would result in wildlife exposure at dietary no effect and low effect concentrations. The MDEQ ERA developed threshold concentrations in soils for the American robin, white-footed and deer mouse, great horned owl, and red fox. The food web model incorporates assumptions regarding the dietary composition of these species and the uptake of PCBs from soil into their food items, which are described in detail by Camp Dresser & McKee (2003b). Table 7.24 presents the soil PCB concentrations that are estimated to produce exposure for the selected wildlife species at

Table 7.24. Site-specific soil PCB threshold concentrations for evaluating injury to selected wildlife species

Receptor species	No effect TRV-based soil threshold (mg/kg dw)	Low effect TRV-based soil threshold (mg/kg dw)
Robin	6.5	8.1
Mouse	21	63
Owl	2.9	8.5
Fox	5.9	29.5

their dietary no effect and low effect values (Camp Dresser & McKee, 2003b). The Trustees use the low effect TRV based soil thresholds as developed by MDEQ to evaluate injury to floodplain soils in this Stage I Assessment.

7.10.3 Results

The combined data sources described in Section 7.10.1 yield 475 surface soil samples from within the KRE floodplain or former impoundment areas. Of these, 222 are located within the boundaries of one of the former impoundments (Plainwell, Otsego, Trowbridge), and 90 are located in the floodplain area of the Otsego City impoundment. An additional 163 samples are located within the floodplain of the Kalamazoo River, but not within the boundaries of any of the four former or current impoundments. Analytical detection limits for the floodplain soil samples included in this discussion range from 0.037 to 0.35 mg/kg.

Former Plainwell impoundment

Surface soil PCB concentrations in the former Plainwell impoundment range from 0.053 to 134 mg/kg. Concentrations in 53% of the samples from within the former impoundment boundary are greater than the injury threshold for robins of 8.1 mg/kg, and 3% are more than 10 times greater (Figure 7.10). Concentrations in 53% of the surface soil samples also exceed the injury threshold for owls of 8.5 mg/kg, and 2% are more than 10 times greater (Figure 7.11). Concentrations in 26% of the surface soil samples from the Plainwell impoundment exceed the fox injury threshold (Figure 7.12). Five percent of the samples have concentrations greater than the mouse threshold (Figure 7.13).

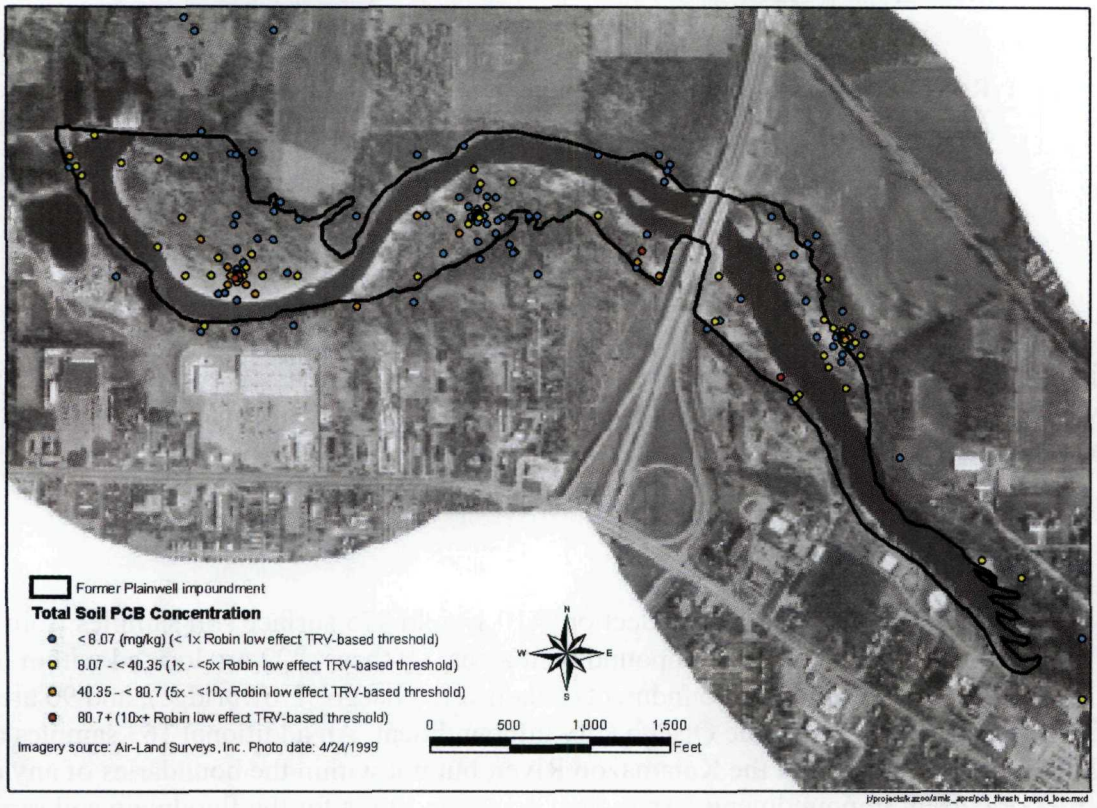

Figure 7.10. Surface soil PCB concentrations in the former Plainwell impoundment compared to the soil threshold for injury to robins. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

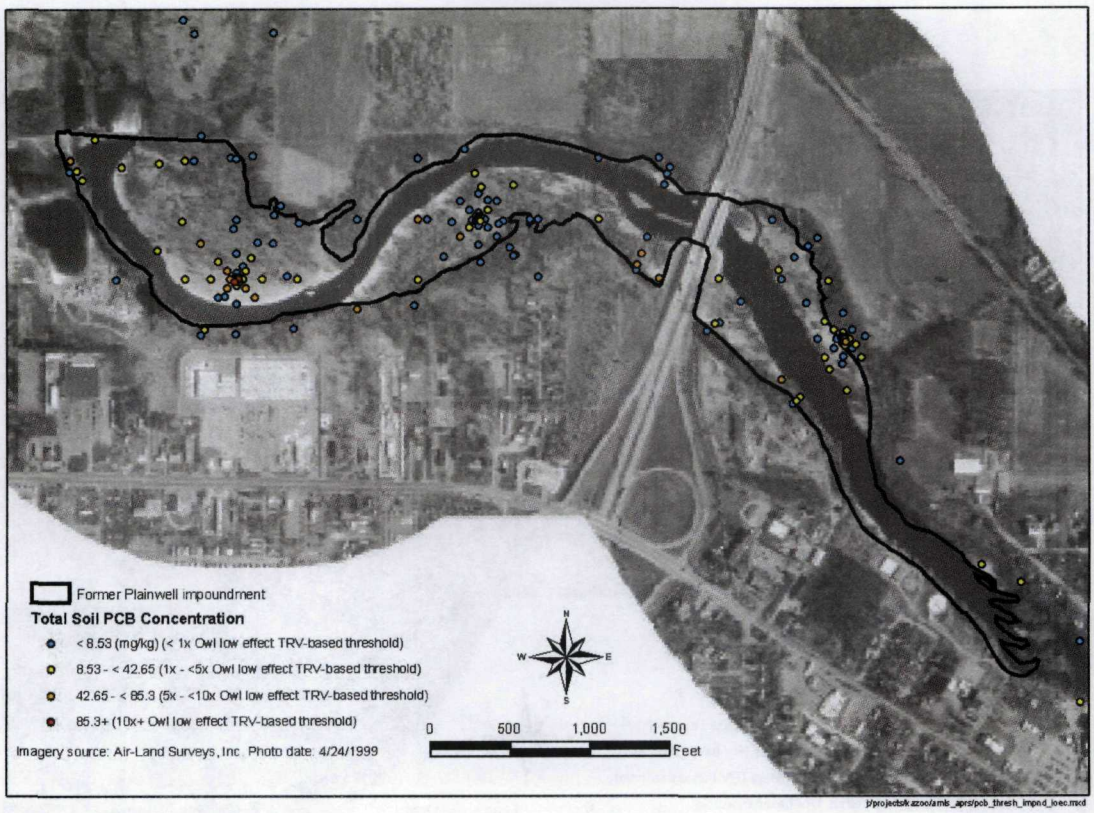

Figure 7.11. Surface soil PCB concentrations in the former Plainwell impoundment compared to the soil threshold for injury to owls. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

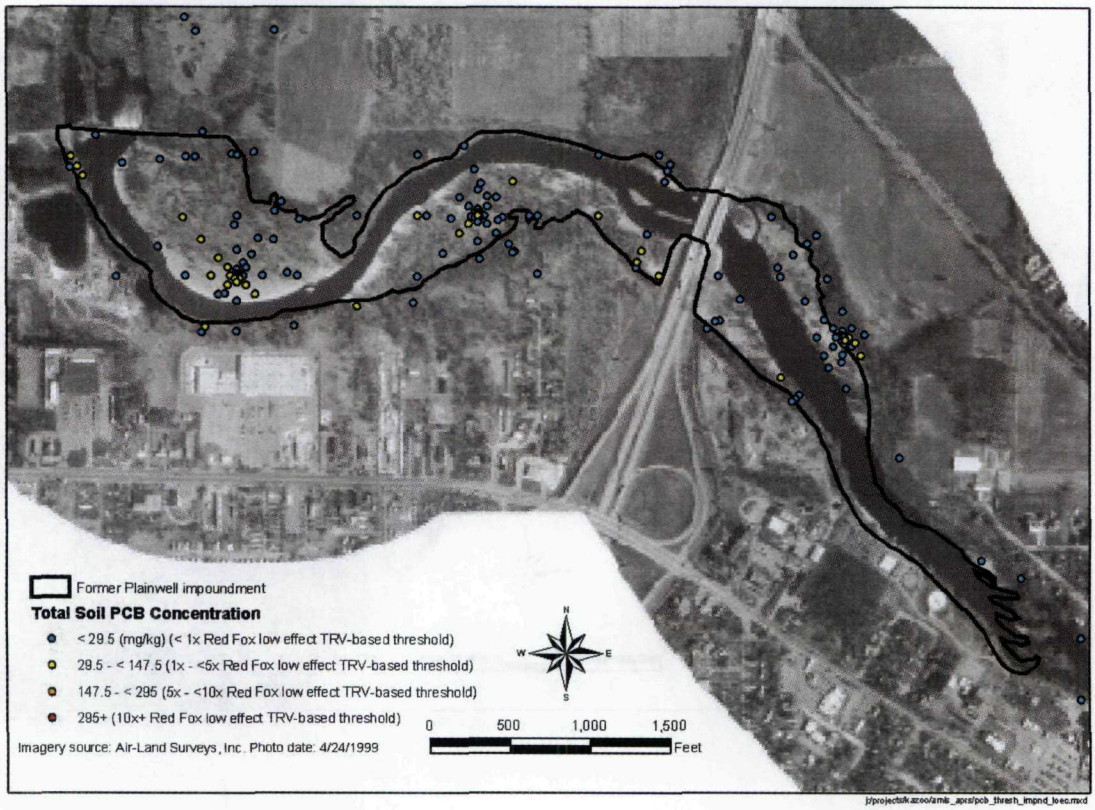

Figure 7.12. Surface soil PCB concentrations in the former Plainwell impoundment compared to the soil threshold for injury to foxes. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

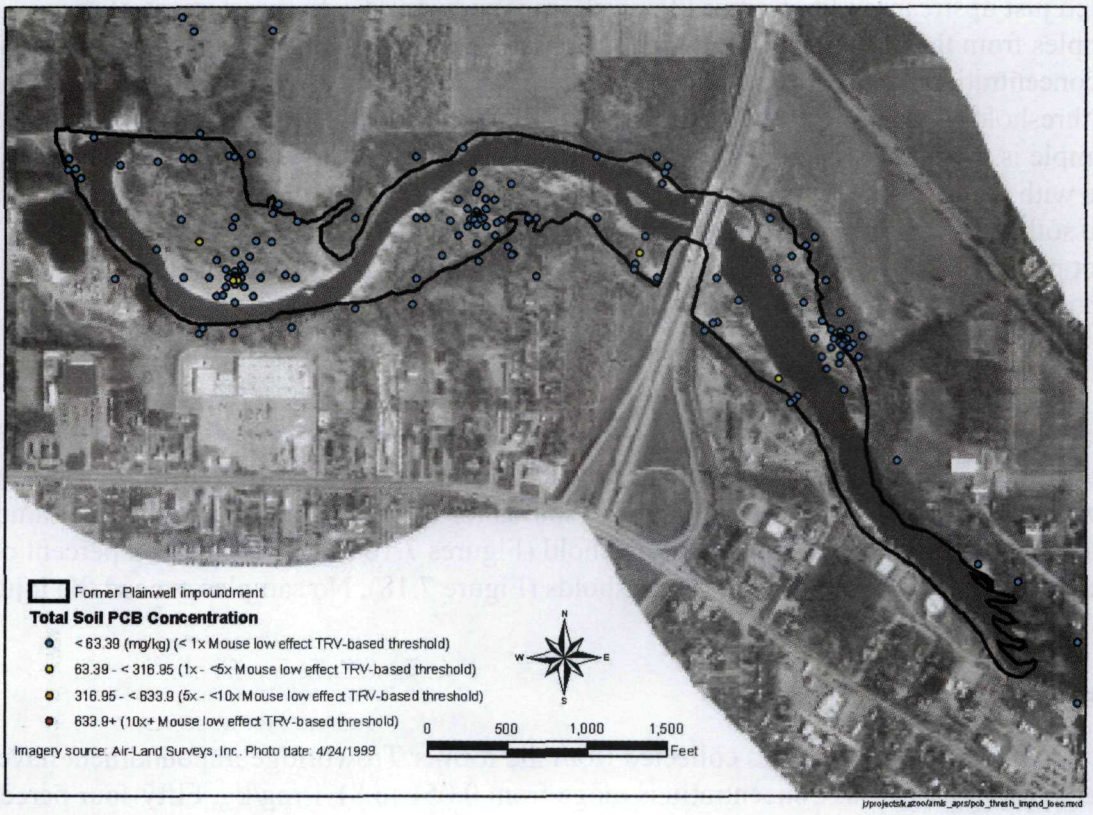

Figure 7.13. Surface soil PCB concentrations in the former Plainwell impoundment compared to the soil threshold for injury to mice. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

Otsego City impoundment

PCB concentrations in surface soil from the Otsego City impoundment area are lower than those observed just upstream in the former Plainwell impoundment. Forty-two percent of the 90 samples from the Otsego City impoundment area have detectable concentrations of PCBs, and those concentrations range from 0.0034 to 58.9 mg/kg. Eight percent of samples exceed the injury threshold for robins, and four percent exceed the injury threshold for owl, although only one sample is more than five times greater than either threshold (Figures 7.14 and 7.15). The sample with the highest concentration, located just upstream of the dam, is the only case where surface soil concentrations in the Otsego City impoundment exceed the injury threshold for red fox (not plotted). None of the surface soil samples collected from around this impoundment have concentrations that are greater than the mouse threshold (not plotted).

Former Otsego impoundment

All but two surface soil samples of the 26 collected from within the former Otsego impoundment have detectable levels of PCBs. Detected concentrations range from 0.13 to 61 mg/kg. Fifty-eight percent of samples exceed the injury thresholds for both robin and owl; only one sample is more than five times greater than either threshold (Figures 7.16 and 7.17). Fifteen percent of samples also exceed the red fox injury thresholds (Figure 7.18). No samples exceed the injury threshold for mouse (not plotted).

Former Trowbridge impoundment

All of the 63 surface soil samples collected from the former Trowbridge impoundment have detectable levels of PCBs. Concentrations range from 0.051 to 81.1 mg/kg. Fifty-four percent of samples from within the impoundment boundary exceed the injury threshold for robins, with 2% more than 10 times greater than this threshold (Figure 7.19). Fifty-two percent of samples have PCB concentrations greater than the owl injury threshold (Figure 7.20), 21% exceed the fox threshold (Figure 7.21), and two samples exceed the mouse threshold (Figure 7.22).

Other Kalamazoo River floodplain soils

Soil samples collected from the Kalamazoo River floodplain outside of impoundment or former impoundment areas generally have lower PCB concentrations than those that were collected in the three former impoundments and in the Otsego City impoundment. Fifty of the 163 surface samples from the floodplain do not have detectable levels of PCBs. Detected concentrations range from 0.026 to 29.3 mg/kg. Two of the surface samples in the Kalamazoo River floodplain are higher than the injury thresholds for robins and owls, both by less than 5 times. No samples exceed the mouse or fox injury thresholds.

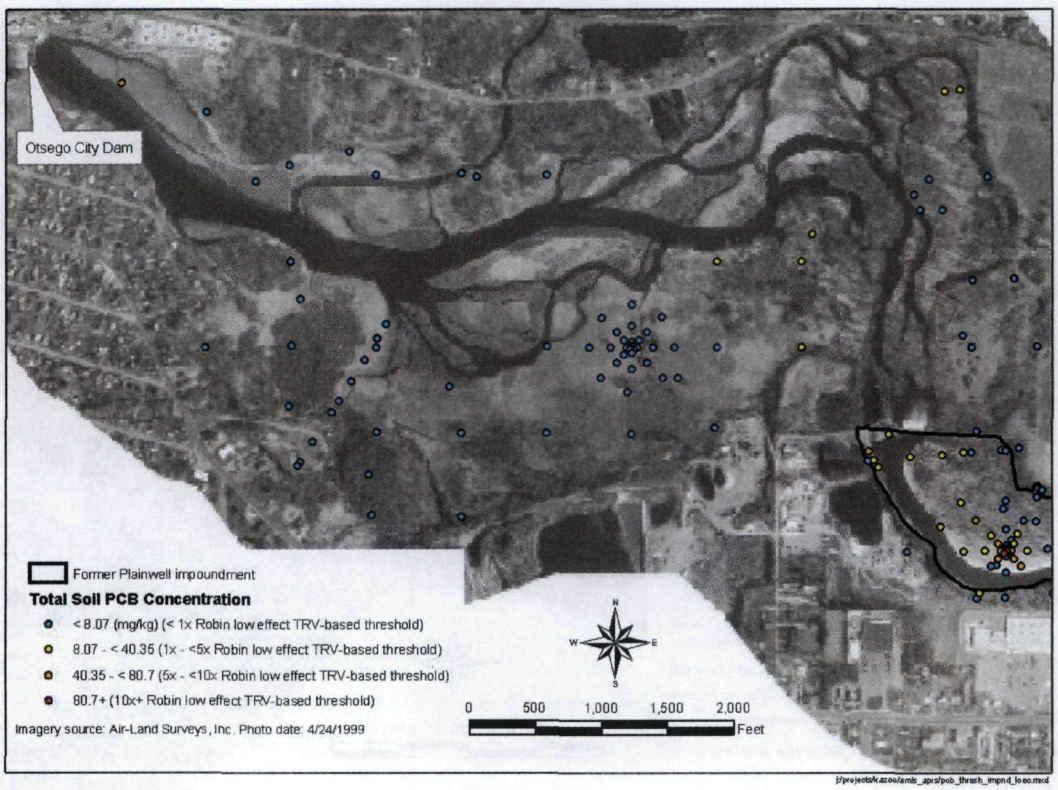

Figure 7.14. Surface soil PCB concentrations in the Otsego City impoundment compared to the soil threshold for injury to robins. No boundary is available for the Otsego City impoundment. Samples located within or upstream of the former Plainwell impoundment boundary in this figure are not included in the description of data in Section 8.3.3.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

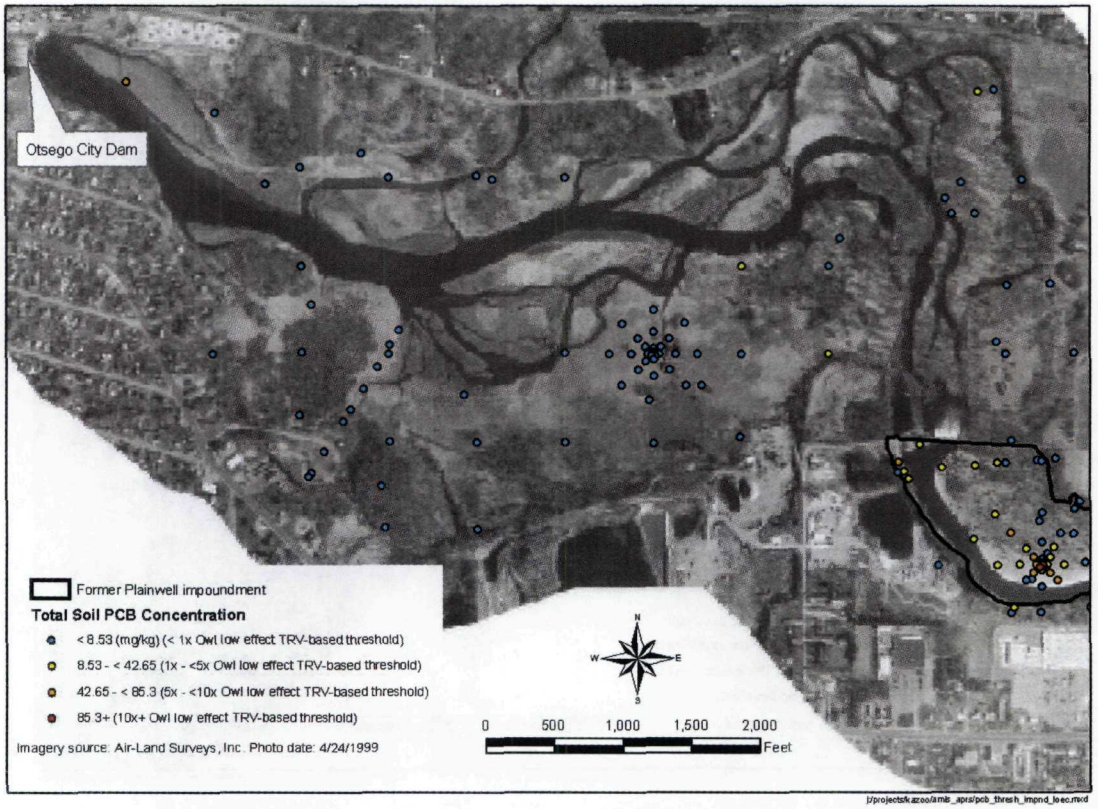

Figure 7.15. Surface soil PCB concentrations in the Otsego City impoundment compared to the soil threshold for injury to owls. No boundary is available for the Otsego City impoundment. Samples located within or upstream of the former Plainwell impoundment boundary in this figure are not included in the description of data in Section 8.3.3.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

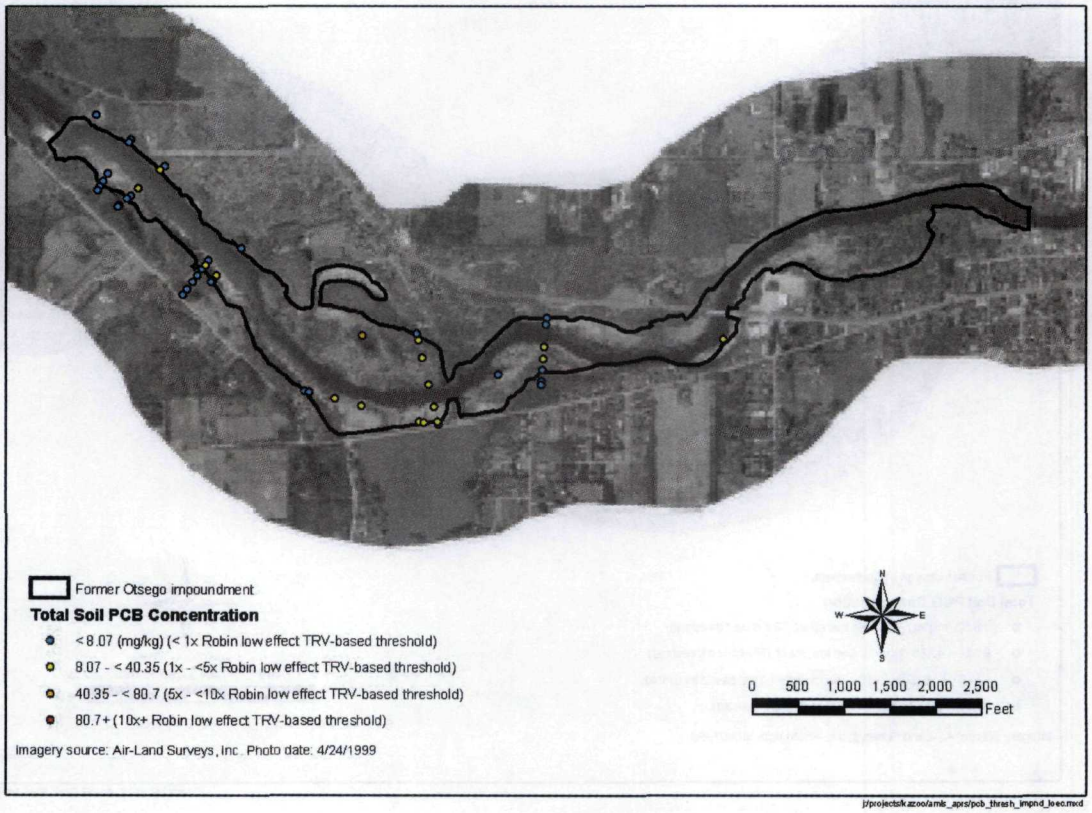

Figure 7.16. Surface soil PCB concentrations in the former Otsego impoundment compared to the soil threshold for injury to robins. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

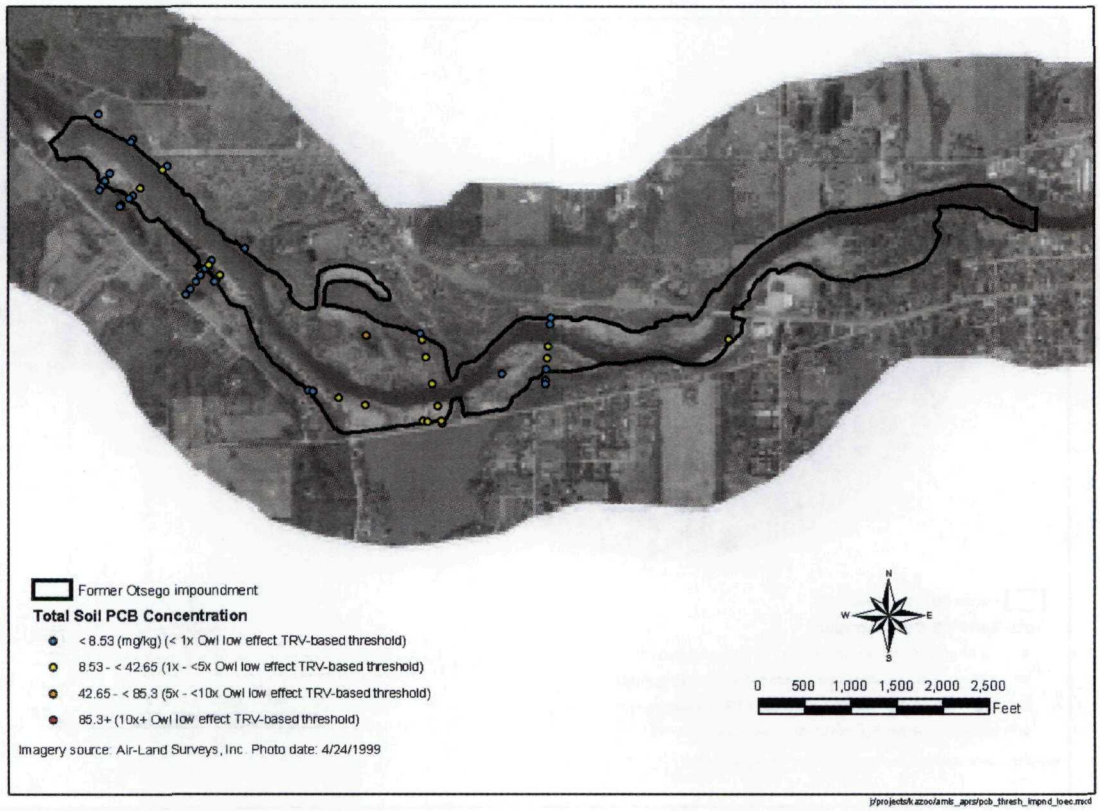

Figure 7.17. Surface soil PCB concentrations in the former Otsego impoundment compared to the soil threshold for injury to owls. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

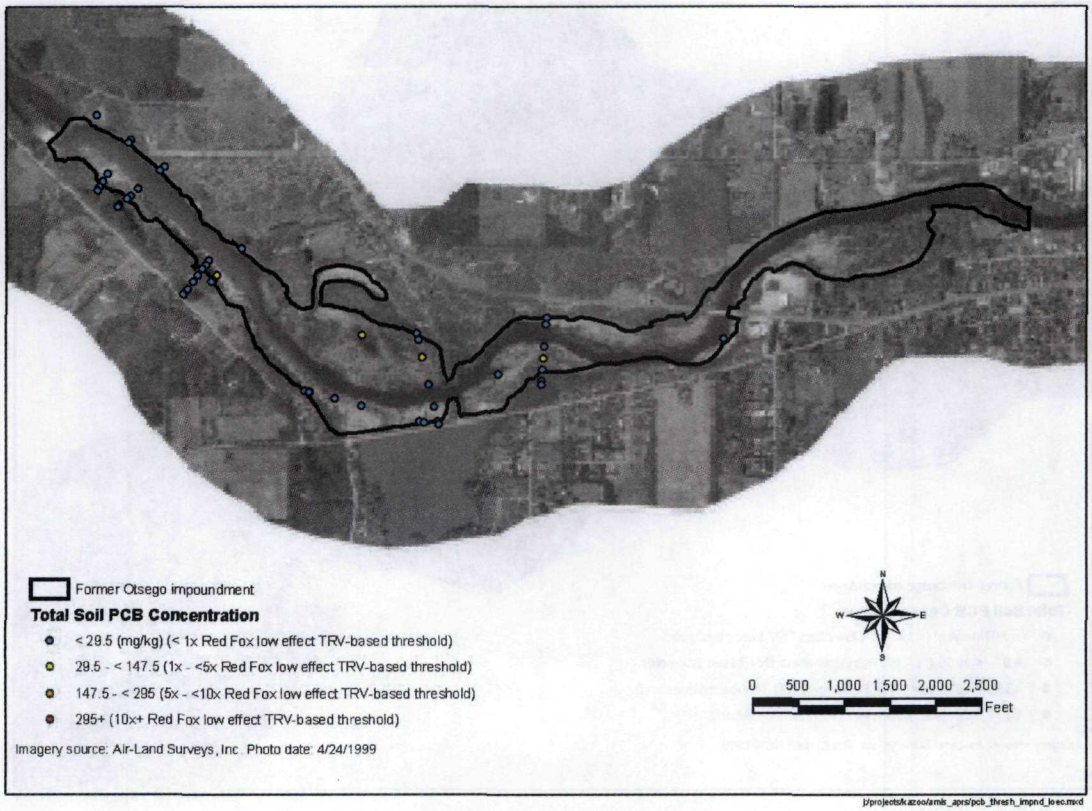

Figure 7.18. Surface soil PCB concentrations in the former Otsego impoundment compared to the soil threshold for injury to foxes. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

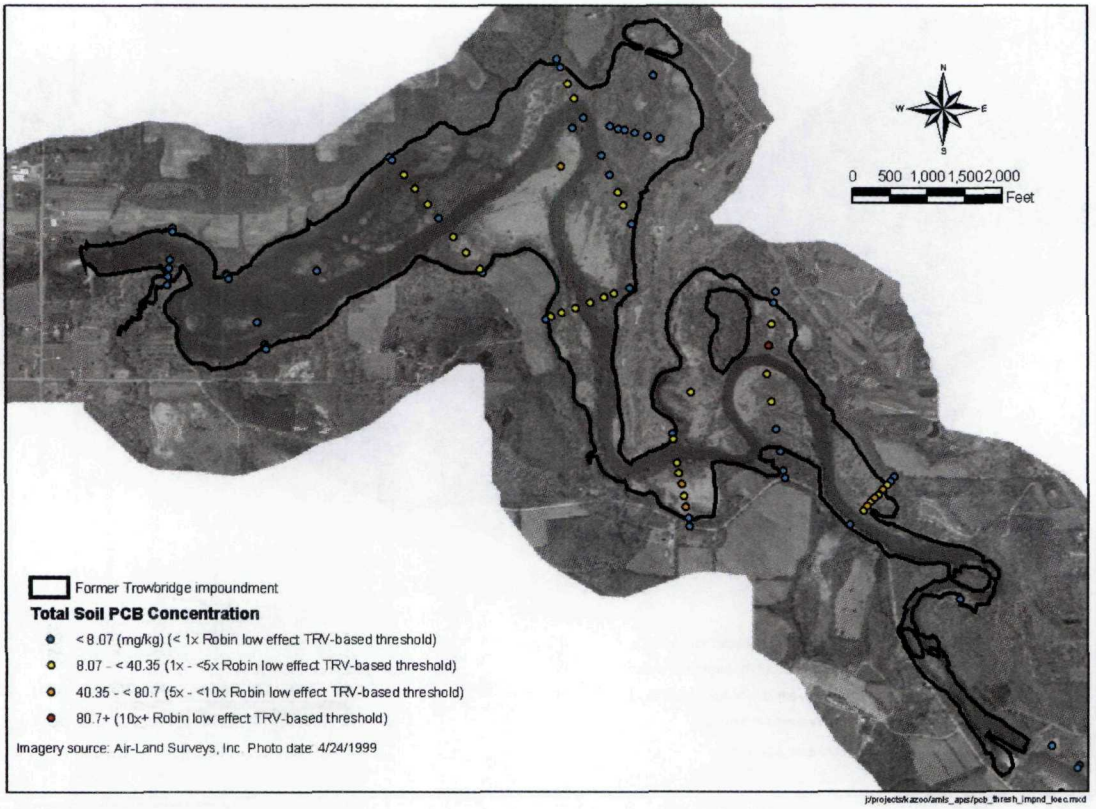

Figure 7.19. Surface soil PCB concentrations in the former Trowbridge impoundment compared to the soil threshold for injury to robins. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

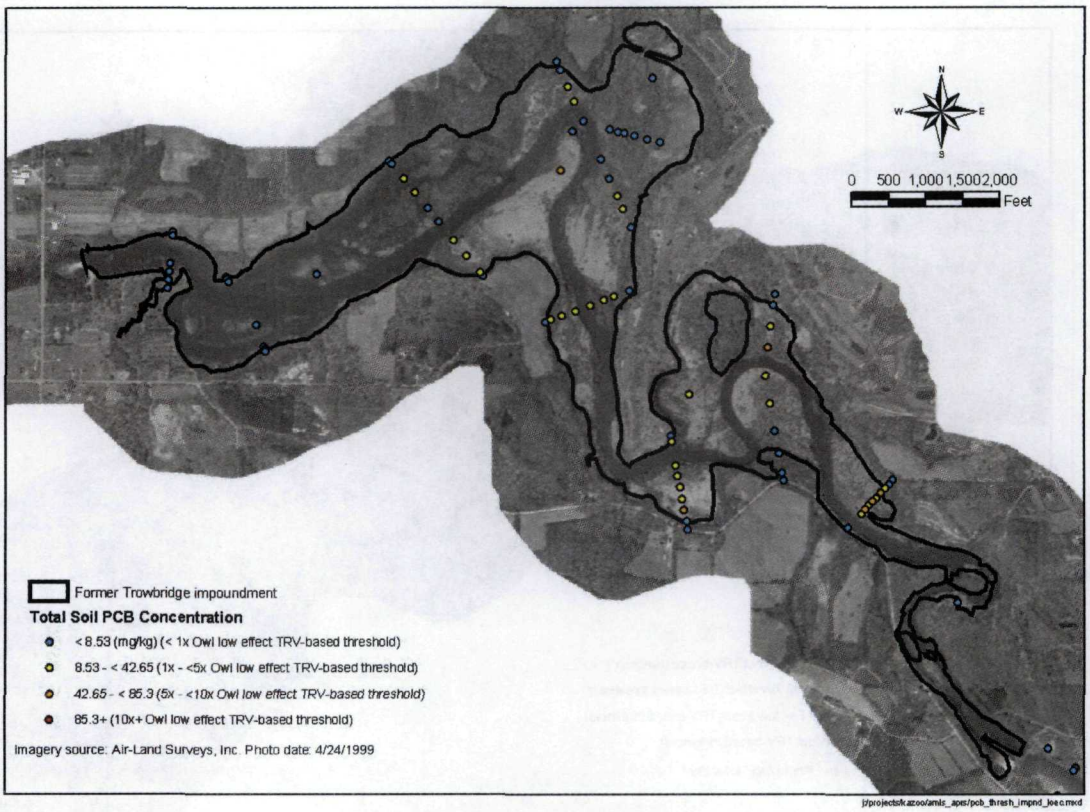

Figure 7.20. Surface soil PCB concentrations in the former Trowbridge impoundment compared to the soil threshold for injury to owls. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

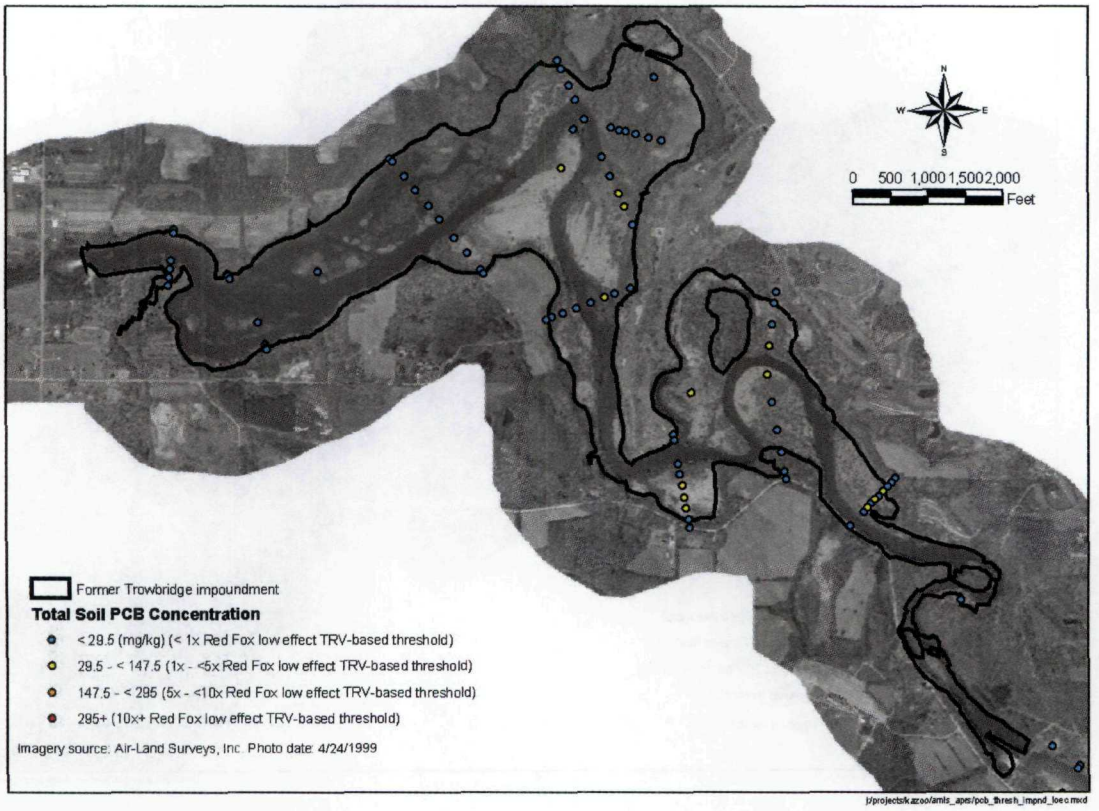

Figure 7.21. Surface soil PCB concentrations in the former Trowbridge impoundment compared to the soil threshold for injury to foxes. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

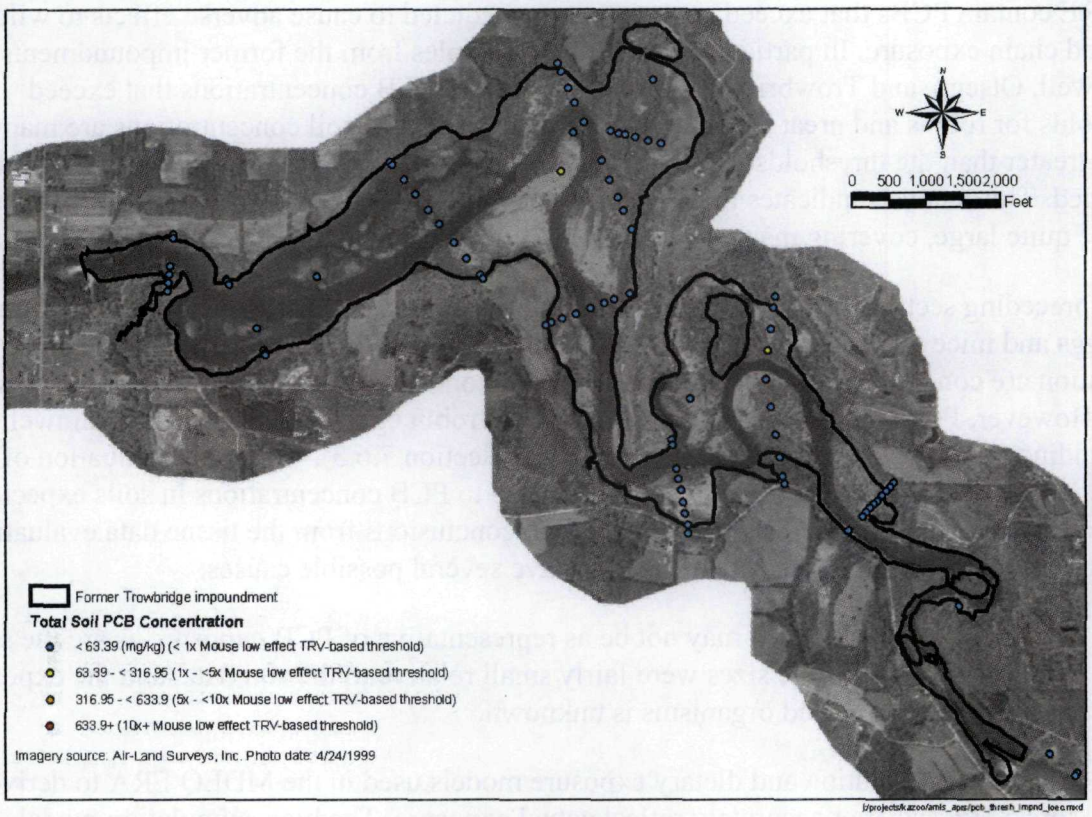

Figure 7.22. Surface soil PCB concentrations in the former Trowbridge impoundment compared to the soil threshold for injury to mice. Points that are located beyond the boundary of the impoundment are not included in analysis, but are plotted for reference.

Sources: Blasland, Bouck & Lee, 2001; Camp Dresser & McKee, 2002a, 2003b.

7.10.4 Conclusions of the floodplain soil evaluation

An evaluation of floodplain surface soil PCB concentrations shows that many floodplain areas of the KRE contain PCBs that exceed concentrations predicted to cause adverse effects to wildlife via food chain exposure. In particular, many of the samples from the former impoundments (Plainwell, Otsego, and Trowbridge) contain surface soil PCB concentrations that exceed thresholds for robins and great horned owls. In many cases, the soil concentrations are many times greater than the thresholds. However, soil thresholds for mice and red fox are only rarely exceeded. This analysis indicates that the spatial extent of injuries to some KRE wildlife species may be quite large, covering many acres of the former impoundments and other floodplain areas.

In the preceding sections of this chapter, PCB concentration data for KRE robin and great horned owl eggs and mice tissue were presented and evaluated. The results of this floodplain soils evaluation are consistent with the evaluation of PCB concentration data for great horned owl and mice. However, PCB concentrations measured in two robin eggs from the former Plainwell impoundment are less than TRVs for egg mortality (Section 7.6.3), while this evaluation of soil PCB concentrations indicates that robins are exposed to PCB concentrations in soils expected to cause adverse effects. The differences between the conclusions from the tissue data evaluation and from the floodplain soil data analysis may have several possible causes:

- The tissue collection data may not be as representative of PCB exposure as are the soil data. The tissue sample sizes were fairly small relative to the soil data, and the exposure history of the collected organisms is unknown.

- The bioaccumulation and dietary exposure models used in the MDEQ ERA to derive soil thresholds may not accurately reflect actual exposure. The bioaccumulation model predicts higher exposure for robins than indicated by the available tissue data. Field data collections are currently being conducted to more fully characterize PCB bioaccumulation in the KRE (Giesy Ecotoxicology, 2001), and the results of these studies may help explain the apparent discrepancies between the currently available field data and the bioaccumulation model.

- A combination of the two factors.

Nevertheless, the analysis of the available floodplain soil PCB data indicates that wildlife species are exposed to PCBs in KRE floodplain areas at concentrations sufficient to cause adverse effects over fairly large areas.

7.11 Conclusions

This chapter presents a Stage I Injury Assessment for KRE wildlife based on available information and data. Two types of injury to wildlife are evaluated: exceedences of the FDA tolerance level for PCBs in waterfowl tissue; and adverse changes in viability resulting from toxicological effects of PCBs.

Available data on PCB concentrations in mallard duck tissue indicate that injury has occurred. The PCB concentration in one of two available mallard duck samples was 6 times higher than the FDA tolerance level. Thus the Trustees conclude that mallard ducks are injured according to the definition in 43 C.F.R. § 11.62 (f)(1)(ii). However, because the data are minimal, the Stage I Assessment does not draw any conclusions about the spatial or temporal extent of this injury.

Table 7.25 summarizes the conclusions of the Stage I Injury Assessment about injuries to KRE wildlife based on adverse changes in viability. The Trustees conclude that bald eagles, piscivorous birds, predatory birds, and mink are injured by exposure to KRE PCBs. Additionally, omnivorous birds (such as robin) are probably exposed to PCBs in KRE floodplain surface soils at concentrations sufficient to cause injury. The severity of the injuries probably ranges from sublethal effects (e.g., reduced chick growth rate, embryo abnormalities) in some piscivorous birds to substantially reduced reproductive success in bald eagles and mink. Based on the available information, the injuries to wildlife most likely occur throughout the KRE, and they have been occurring for several decades (probably beginning soon after the PCB releases into the KRE began).

KRE bald eagle data support the conclusion that eagles are injured by exposure to PCBs. Bald eagle reproductive rates are lower near the Kalamazoo River than in other coastal and inland Michigan sites. Although nests have been attempted since 1990, the first offspring were not successfully reared until 1998, and the average nesting success between 1990 and 2003 was only 0.2 young per nest, far below normal replacement rates. Bald eagle eggs and plasma collected in the KRE have total PCB concentrations sufficient to substantially reduce reproductive rates. Furthermore, PCB concentrations in fish, an important bald eagle food source, exceed dietary TRVs for toxicological effects. The ERA conducted by the MDEQ also concluded that bald eagles are at high risk from exposure to PCBs. Therefore, the Trustees conclude that KRE bald eagles are injured by exposure to PCBs, and that the injury is increased egg mortality and decreased reproductive success.

Table 7.25. Summary of Stage I Assessment conclusions regarding toxicological injuries to wildlife

Species/resource	Injured?[a]	Nature of injury	Basis for conclusions	Probable spatial extent of injury
Bald eagle	Yes	Egg mortality, reduced productivity	Fish [PCB] > dietary TRVs Productivity data Egg [PCB] > TRVs Plasma [PCB] > TRVs	Two breeding areas in Allegan State Game Area, and one near New Richmond. Depending on suitability of habitat, rest of river downstream of PRP facilities.
Piscivorous birds	Yes	Sublethal effects Reproductive effects in sensitive species Egg mortality	Fish [PCB] > dietary TRVs MDEQ ERA results Egg [PCB] > TRVs (e.g., great blue heron)	Entire river downstream of PRP facilities (based on fish [PCB]).
Predatory birds (e.g., great horned owl, red-tailed hawk)	Yes	Egg mortality Reproductive impairment	Egg [PCB] > TRVs MDEQ ERA results for great horned owl Floodplain soil PCB concentrations > great horned owl toxicity thresholds (some > 10 times)	Floodplains soils in former impoundments and other areas (based on exceedences of great horned owl thresholds). Egg data available only for Allegan State Game Area.
Passerine birds (e.g., robin, red-winged blackbird, wood thrush, yellow warbler) and wood duck	Possible	Reproductive impairment	Egg [PCB] ≅ sensitive species TRVs MDEQ ERA results for robins Floodplain soil PCB concentrations > robin toxicity thresholds (some > 10 times)	Floodplains soils in former impoundments and other areas (based on exceedences of robin thresholds).
Waterfowl (e.g., mute swan)	No	-	Egg [PCB] < TRVs	-
Mink	Yes	Reproductive impairment	Fish [PCB] > dietary TRVs Mink whole body and liver [PCB] > TRVs MDEQ ERA results Anecdotal trapping success data	Entire river downstream of PRP facilities.

Table 7.25. Summary of Stage I Assessment conclusions regarding toxicological injuries to wildlife (cont.)

Species/resource	Injured?[a]	Nature of injury	Basis for conclusions	Probable spatial extent of injury
Red fox	No	Reproductive impairment	MDEQ ERA concludes risk for red fox unlikely unless diet consists of prey which have substantial amounts of PCBs Floodplain soil PCB concentrations > red fox toxicity thresholds	Exceedences occur within limited areas within floodplains soils and former impoundments and other areas.
White-footed/deer mouse	No	-	Mouse whole-body [PCB] < TRVs Floodplain soil PCB concentrations < toxicity thresholds (few exceedences) MDEQ ERA results	-
Muskrat	No	-	Muskrat liver [PCB] < TRVs Muskrat whole-body [PCB] < TRVs MDEQ ERA results	-

a. Yes = more likely than not that PCB exposure is at least a contributing factor to adverse changes in organism viability. Possible = some evidence for injuries, but important uncertainties exist. No = evidence for no injury.

Other piscivorous bird species are also exposed to elevated dietary PCB concentrations. Many of the PCB concentration measurements in whole body fish from the KRE exceed dietary toxicological benchmarks for causing sublethal effects in birds, and some exceed concentrations shown to cause embryomortality in species that are sensitive to PCBs. PCBs have also been measured in KRE great blue heron eggs at concentrations greater than thresholds for reduced hatching success. Although the PCB sensitivity of KRE piscivorous bird species is unknown, the available data support the conclusion that birds that consume fish from the KRE, such as the great blue heron or belted kingfisher, are injured from exposure to PCBs. Based on the spatial extent of PCB contamination in KRE fish, the injury is expected to occur throughout the Kalamazoo River and Portage Creek downstream of PRP facilities.

There are limited data on the PCB exposure of predatory birds in the KRE. PCB concentrations measured in great horned owl and red-tailed hawk eggs fall within the range expected to cause reduced egg hatching success. Additionally, the concentration of TCDD-eqs from PCBs in great

horned owl eggs falls within the range associated with embryomortality effects in sensitive species. Although the data are limited, they support the conclusion that predatory birds are exposed to PCBs at concentrations sufficient to cause toxicological effects. The ERA conducted by the MDEQ also concluded that great horned owls are at high risk from exposure to PCBs. Therefore, the Trustees conclude that at least some predatory birds of the KRE are injured.

Egg PCB concentration data for many species of passerine birds (robin, red-winged blackbird, tree swallow, wood thrush, yellow warbler) and waterfowl (great blue heron, mute swan and wood duck) are less than threshold ranges for toxicological effects for tolerant species, but are within or greater than ranges for sensitive species. Egg PCB concentrations in eastern bluebird and house wren eggs also fall within the range for embryomortality in more tolerant species. The sensitivities of the species collected are not known. Therefore, these data indicate that these bird species may be injured by their exposure to KRE PCBs, depending on their sensitivity to PCB toxicity. In contrast, an evaluation of PCB concentrations in floodplain surface soils using the soil threshold concentrations developed in the MDEQ ERA food web model predicts that omnivorous birds such as robins are exposed to PCBs at concentrations sufficient to cause reproductive impairment. Soils in the former impoundments and other floodplain areas contain PCBs that are many times higher than soil thresholds for food chain effects to robins. The discrepancy between the predictions of the bioaccumulation model and the observed robin egg PCB data may be caused by a lack of representativeness of the available robin egg data, inaccuracies in the model, or a combination of both. Therefore, the Trustees conclude in this Stage I Injury Assessment that passerine species are possibly injured by exposure to PCBs.

Several types of data support the conclusion that KRE mink are injured by exposure to PCBs. PCB concentrations in fish from throughout the KRE exceed dietary thresholds for adverse reproductive effects in mink. Measured PCB concentrations in mink carcasses and livers captured from the KRE exceed thresholds for the same effects. Anecdotal trapping information suggests that mink populations along the Kalamazoo River downstream of PRP facilities are suppressed. The MDEQ ERA concludes that mink are at high risk from PCB exposure. Therefore, the Trustees conclude that mink are injured by exposure to KRE PCBs.

Available data indicate that KRE red fox are unlikely to be injured. The MDEQ ERA also concluded red fox are at low risk from PCB exposure, unless foraging is concentrated in riparian areas and the diet consists largely of organisms that have accumulated high concentrations of PCBs. Floodplain soils in some areas of the KRE contain PCBs at concentrations predicted to cause toxicity (based on the MDEQ ERA), but exceedences are infrequent.

Available data indicate that muskrat and mice are not injured. Measured PCB concentrations in KRE muskrat livers and whole-body samples are less than TRVs for toxic effects. The MDEQ ERA also concluded that these organisms are at low risk from PCB exposure.

Finally, the wildlife injury conclusions of the Stage I Assessment are limited to the species, resources, and areas for which relevant data are available. Bald eagles and mink are two key species in the assessment, and ample injury data and information are available for these species. However, PCB exposure data are available for only a small subset of the bird and mammal species present in the KRE, and only the bald eagle has been studied for information on actual adverse effects in the field. Therefore, the extent of injuries to wildlife species in the KRE may be greater than indicated by the available data.

8. Indirect Injuries

8.1 Introduction

Section 107(f)(1) of CERCLA provides broad authority to natural resource trustees to recover damages to restore, replace, or acquire the equivalent of natural resources in the case of injury to, destruction of, or loss of natural resources [42 U.S.C. 9607(f)(1)]. The DOI regulations at 43 C.F.R. Part 11 elaborate on what damages may be recovered, including indirect injuries to natural resources.

43 C.F.R. § 11.15(a)(1) states that trustees may recover damages resulting from natural resource injuries that are reasonably unavoidable as a result of response actions taken or anticipated. Furthermore, 43 C.F.R. § 11.14(e) defines "baseline" as the condition or conditions that would have existed at the assessment area had the release of the hazardous substance under investigation not occurred. The absence of responsible parties' actions, and not simply the absence of a particular chemical, defines baseline. Therefore, when a responsible party's releases require response actions or changes in resource management that cause injuries, natural resource trustees may recover damages for those injuries. These injuries that result from response actions are referred to as an indirect injuries, and they are the subject of this chapter.

This approach is consistent with the requirements for quantifying injuries and damages under the DOI regulations. For instance, 43 C.F.R. § 11.72(b)(1) states that baseline data should reflect conditions that would have been expected at the assessment area had the release of hazardous substances in question not occurred, taking into account both natural processes and those that are the result of human activities. Further, 43 C.F.R. § 11.82(b)(1)(i) states that restoration or rehabilitation actions are in addition to response actions completed or anticipated pursuant to the National Contingency Plan. In addition, 43 C.F.R. § 11.82(d) specifies that the results of any actual or planned response actions, and the potential for additional injury from such actions, including long-term and indirect impacts, are factors to consider when selecting a restoration alternative. Finally, 43 C.F.R. § 11.84(c)(2) states that damages are the residual to be determined by incorporating the effects, or anticipated effects, of any response actions.

Therefore, the Trustees include an analysis of injuries that may result from response actions necessary to address hazardous substance releases by responsible parties. Section 8.2 outlines the trustees' approach. Then, Sections 8.3 through 8.8 describe potential indirect injuries that may result from a variety of potentially necessary response actions. Finally, Section 8.9 presents the Trustees' conclusions about indirect injuries.

8.2 Stage I Assessment Approach

The nature and extent of indirect injuries that result from response actions to address PCB contamination at the site vary depending on the type of response actions that will be selected and implemented. At this time, the RI/FS for the site is ongoing and the response actions to address PCB contamination have not yet been selected, so the indirect injuries that may result from response actions cannot yet be identified. However, a document prepared by the PRPs (Blasland, Bouck & Lee, 2000a) provides some insight on the types of response actions that are likely to be considered during the RI/FS process. Table 8.1 lists the kinds of remedial options that may be implemented at the site, based on the options listed in the PRP document. This chapter presents a discussion of the possible nature of indirect injuries that may be associated with each of the potential response actions. As more becomes known regarding the probable response actions that will be implemented at the site, the Trustees will continue to evaluate the indirect injuries that will result from the actions.

Table 8.1. Remedial options for the Kalamazoo River

Remedial option	Description	Chapter section
No further action	No further remedial activities to address PCB contamination	8.3
Access/deed restrictions	Constraints such as fencing and signs to limit access to property, and restrictions on future river use	8.4
Bank stabilization	Placement of vegetation or riprap material along banks of exposed sediment areas	8.5
Pool elevation control	Maintenance of existing dams to minimize downstream transport of sediment	8.6
Sediment capping	Covering of instream sediments with materials such as clean sediment, sand, gravel, geotextiles, or other capping materials	8.7
Sediment removal and disposal	Removal of instream sediments using mechanical or hydraulic methods (may include temporary alteration of channel) and disposal in existing or newly constructed landfill	8.8

Source: Blasland, Bouck & Lee, 2000a; options extracted from Table 3.2.

8.3 No Further Action

The no further action option must be included in the analysis of remedial action alternatives. Under the no further action option, no additional measures would be conducted to address PCB contamination at the site. Therefore, future direct injuries from ongoing PCB contamination will be the greatest under this option. Furthermore, if no additional work to reconstruct and maintain

the existing dam sills at the former Plainwell, Otsego, and Trowbridge impoundments is conducted, the structures are likely to fail (Hanshue, 2002). If this happens, large quantities of the PCB contaminated sediments that have accumulated behind the structures would be released downstream, causing a substantial increase in the exposure of natural resources to PCBs. This would result in an even higher level of direct PCB injuries in the KRE.

If, however, the dam structures are kept in place as a means of containing the PCB contaminated sediment behind them, then indirect injuries will result from the river not being returned to a more natural, free-flowing state. These indirect injuries are discussed in Section 8.6.

8.4 Access/Deed Restrictions

Access restrictions could be used to limit public entry into areas used for fishing, thereby limiting consumption of PCB contaminated fish from the river, and deed restrictions could be used to limit activities on private lands to reduce potential PCB exposure (Blasland, Bouck & Lee, 2000a). Such restrictions may result in decreases of human use services provided by the KRE. For example, recreational fishing in PCB contaminated areas provides some positive value to anglers, even though the value is less than the value without any PCB contamination (Breffle et al., 1999). Therefore, damages associated with reductions in human use services that result from future access or deed restrictions may be quantified by the Trustees if access or deed restrictions are part of the selected response action for the site.

8.5 Bank Stabilization

Stabilizing the banks of the Kalamazoo River in former impoundment areas could be used to reduce bank erosion and the ongoing PCB load from the banks into the river (Blasland, Bouck & Lee, 2000a). However, bank stabilization can also lead to river channelization and prevent natural geomorphologic processes from operating, thereby causing indirect injuries. The engineering method selected for bank stabilization will affect the nature and level of indirect injury resulting from this alternative. Use of a technology such as riprap (Figure 8.1) or sheetpile (Figure 8.2) will essentially eliminate riparian and nearshore aquatic habitat, alter the hydrologic and temperature regimes of the river, and affect sediment transport processes. Additionally, this type of engineered shoreline may have effects on the human use of the river by altering the aesthetic qualities and/or public access. For example, although ease of access to the shoreline may be facilitated by bank stabilization, human use may be impacted because of the absence of natural vegetation and the variety of habitats that provide scenic value to the shoreline. It is also likely that the quality of birdwatching and fishing will be reduced due to a loss of shoreline vegetation and nearshore instream habitat types.

Figure 8.1. Shoreline stabilized with riprap.

Source: Wyoming Natural Resources Conservation Service, 2002.

Figure 8.2. Sheetpile along the Kalamazoo River.

Source: MDEQ, 1997c.

The key to functioning stream systems is their dynamic nature, both seasonally and spatially. Completion of the life cycle of many riverine species requires an array of different habitat types whose seasonal availability is determined by the flow regime. Because overall stream behavior is maintained in dynamic equilibrium, changes in one of several variables such as flow, velocity, or streambed substrate will result in compensating changes in the other variables. Channelization and armoring (rip-rapping) of a stream section will cause permanent changes, removing the dynamic nature of not only the altered section, but also affecting the adjoining stream sections further downstream than might initially be anticipated. Stream channelization often increases stream velocity, thereby increasing the erosive power of the stream. Durable protection or armoring is then required to ensure the stability of the engineered modification through all flow events. Over time, maintenance of such modifications may be significant, particularly as other instream structures, such as dams, are modified or removed. The construction phase of such instream modifications alone will cause significant disturbance that will likely increase runoff, sediment transport, and turbidity and reduce downstream water and habitat quality for aquatic life.

Therefore, the intended benefits of channelization and armoring can be accompanied by ecological losses resulting from increased stream velocities and reduced habitat diversity. Instream modifications of this type result in less habitat for organisms living both in or on stream sediments (macroinvertebrates) and for those living in the water (fish). Stream channelization can disrupt riffle and pool complexes needed at different times in the life cycle of certain aquatic organisms and can cause velocity or habitat fragmentation barriers to movement (Federal Interagency Stream Restoration Working Group, 1998). Habitat is lost when large woody debris and undercut banks are removed, as both frequently support a higher density of aquatic macroinvertebrates and provide important feeding, resting and cover areas for fish.

Losses in riparian vegetation are also inevitable with channelization and armoring of streambanks. Riparian vegetation is critically important in stabilizing streambanks, maintaining temperature stability (minimizing rise in summer and minimizing drop in winter), providing microhabitats important to aquatic and terrestrial life, and providing continuous habitat to connect populations of wildlife along the river corridor. Riparian vegetation also provides a source for continual inputs of woody debris and detritus which provide cover and nutritive inputs for sustaining productivity (i.e., food for the base of the aquatic food chain). Such habitat losses can occur through direct removals during construction/modification, or indirectly via increased velocities that flush materials down stream. The net result is reduced habitat, reduced diversity in habitat, and reduced productivity and diversity of aquatic life at all trophic levels from macroinvertebrates to fish.

8.6 Maintaining Dam Structures

Flow of the Kalamazoo River is controlled by a series of dams, some of which have had their superstructures removed to their sill levels (see Figure 1.1). The Plainwell, Otsego, and Trowbridge dams were constructed in the early 1900s for power generation, and have been out of service since 1965 (Blasland, Bouck & Lee, 2000b; Rheaume et al., 2002). Since then, the dams have been removed to their sill levels and allowed to drain. Some sediment was transported downstream by the resulting increase in stream flow velocity, but a great deal of PCB contaminated sediment remains stored behind these dams. One potential remedial alternative is to maintain these dams at sill level to prevent further disturbances and downstream transport of deposited sediment (Blasland Bouck & Lee, 2000a). Complete reconstruction and perpetual maintenance of the structures would be required if maintaining these structures is included as part of a selected PCB remedy for the site.

Were it not for the PCBs released from PRP facilities into the KRE, the State of Michigan would already have removed the bases of the dams at the Plainwell, Otsego, and Trowbridge impoundments (Hanshue, 2002; Sygo, 2002). The MDNR purchased the dams and surrounding areas with the objective to improve the water quality of the Kalamazoo River, and intended to remove the dams to further this goal. However, the State has been unable to remove the dams because of the presence of PCB contaminated wastes behind these dams (Hanshue, 2002). The State has implemented interim measures to stabilize the dams, but does not believe that the dams can be repaired to keep them safely in place for the long term and would not support a remediation scenario that includes dam reconstruction (Hanshue, 2002; Sygo, 2002). Documentation of the State's intent to improve water flow characteristics of the Kalamazoo River goes back at least to 1981 (MDNR, 1981a).

The DOI NRDA regulations define baseline as "the conditions that would have existed at the assessment area had the . . . release of the hazardous substance under investigation not occurred" [43 C.F.R. §11.14(f)]. Therefore, baseline conditions in the KRE include the river with the dam structures removed or capable of being removed without regard to PCB contamination. Adverse impacts caused by the ongoing presence of the dam structures are indirect injuries that result from the PCB releases.

The structures also prevent natural geomorphologic processes from acting on the river, which in turn help establish and maintain fish and wildlife habitat along the river corridor. Finally, human use of the KRE is reduced due to restrictions on boating use because of the structures. The dam structures are impassable to boats or canoes in either direction, and therefore recreational boating use is impaired because of the dams. Overall, the presence of the dams also restricts the State in managing state fisheries and wildlife resources. Maintenance of unwanted and obsolete dams, which degrade the natural resources of the Kalamazoo River, is in direct opposition to the goals of the State (MDNR, 2002; Sygo, 2002). The dam structures have had and will continue to have

several adverse impacts on KRE natural resources. The structures prevent the natural movement of fish and other aquatic biota and can have long-term impacts on the fish assemblages in rivers (Quinn and Kwak, 2003). Additionally, the low velocity of water in impoundments is detrimental to mussels (Mulcrone and Mehne, 2001). In a survey conducted in 2000, Mulcrone and Mehne (2001) found reduced mussel populations in the former Otsego, former Trowbridge, Allegan City, and Lake Allegan impoundments. Catch-per-unit effort ratios in these impoundments were 2.8, 1.6, 0, and 0 mussels/person-hour, respectively. The catch-per-unit-effort in the former Plainwell impoundment was much higher (20.1 mussels/person-hour), and Mulcrone and Mehne (2001) concluded that the current was most likely sufficient to support mussels.

In summary, despite the intent of the State to remove the dam structures to enhance the resources and human uses of the KRE, their removal has been prevented because of the presence of the PCBs in the sediments behind the structures. Therefore, indirect injuries have already occurred. These indirect injuries include the loss of the ecological and human use services that would be present if the river were returned to a more natural, free-flowing state by removal of the dam structures. Indirect injuries associated with the dam structures will continue in perpetuity should the PCB cleanup action include a component that relies upon maintaining the dams as a means of containing PCB contaminated sediments.

8.7 Sediment Capping

Capping of stream sediments may be used to isolate PCB contaminated sediments from the active river system (Blasland Bouck & Lee, 2001) (Figure 8.3). Engineering technologies can enhance this process by increasing the rate of sediment deposition or effectively sealing the sediments. Particle broadcasting introduces additional fine particles to the system to increase the deposition rate, while an engineered cap involves placing one or more layers of material such as sand, gravel or geotextiles over the sediment.

In the short to intermediate time frame, sediment capping in river systems such as the Kalamazoo River can cause impacts to the benthic invertebrate community, with potential implications for fish and associated recreational uses. Assuming that the cap material is eventually covered with natural river sediment, these effects will eventually subside. Therefore, some indirect injuries resulting from engineered sediment caps may be relatively short lived. However, some indirect impacts may last longer. For example, the construction of an in-situ cap may reduce the water depth in the river, and may physically restrict recreational navigation services (Wisconsin Department of Natural Resources, 2003).

Figure 8.3. Cross section of sediment cap over contaminated sediment. An orange worm living in the sediment is visible.

Source: U.S. EPA, 2001.

Concern also remains regarding the long-term effectiveness of caps at isolating sediment contaminants (Palermo et al., 1998). Cap integrity can be compromised by erosion or human activities. Contaminants may migrate up through the cap via advection, diffusion, or bioturbation. In addition, physical and hydraulic characteristics and the existing and future uses of the river will affect the effectiveness of the cap. Ideal locations for in-situ capping are stable sheltered areas with mild currents and either deep water or shallower water which is not exposed to erosion (Wisconsin Department of Natural Resources, 2003). The Kalamazoo River is a dynamic system with a braided channel, and changes in channel location and velocity could compromise the ability of a sediment cap to isolate PCBs in the long term. To the extent that engineered caps do not provide long-term effectiveness in reducing PCB exposure, these impacts would constitute residual direct injuries resulting from the PCBs themselves, rather than indirect injuries.

8.8 Sediment Removal and Disposal

Removal of PCB contaminated sediment from the site has also been proposed as a remedial alternative (Blasland, Bouck & Lee, 2000a). Excavation could be done with a mechanical dredge (Figure 8.4), which physically scoops the sediment from the bottom; a hydraulic dredge, which pumps the sediment in a slurry (Figure 8.5); or by controlling the flow of the river so that sediments can be excavated "in the dry." The removal of PCB contaminated sediments from the KRE can be a very effective strategy for reducing the exposure of natural resources to PCBs, and thereby the ongoing direct injuries to resources. Nevertheless, sediment removal can cause indirect resource injuries that the Trustees may consider in future stages of the NRDA, if sediment removal is selected as a remedial option.

Figure 8.4. Mechanical dredge removing sediment.

Source: NOAA, 2000.

Sediment removal can cause substantial alterations to the river bottom habitat (Cushing, 1999) and associated impacts to benthic communities. For example, Mulcrone and Mehne (2001) reviewed the impacts of dredging activities on mussel populations in the Kalamazoo River. The catch-per-unit-effort at locations where sediment has been removed, the A-Site/Willow Blvd. location (4.5 mussels/person-hour) and the King Highway location (6.9 mussels/person-hour), was lower than the catch-per-unit-effort at a reference location in Battle Creek (10 mussels/ person-hour). These results suggested that mussel populations were affected by dredging activity possibly via physical destruction or shifting of mussel beds.

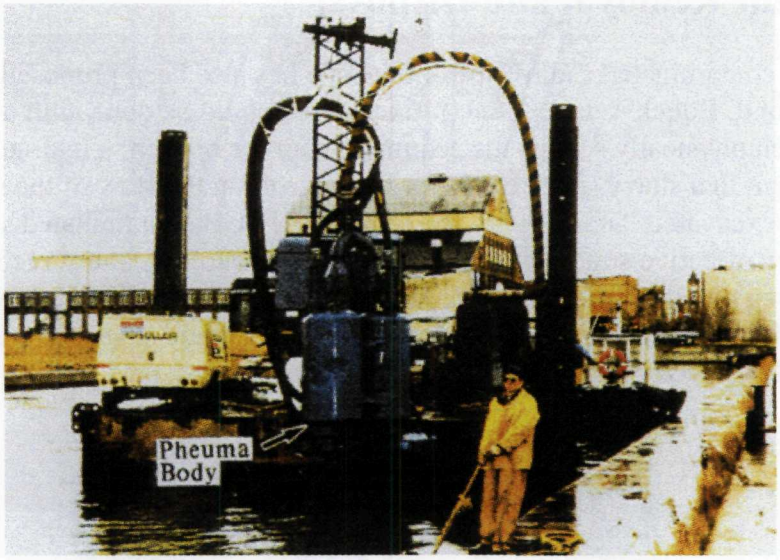

Figure 8.5. Suction-style hydraulic dredge.

Source: Environment Canada, 1996.

As with sediment capping, these impacts should last only over short to intermediate time frames as the areas becomes covered with natural sediment and recolonized. Some development or road construction in the riparian corridor may also be required to provide access to the river, potentially reducing or eliminating areas of riparian habitat.

8.9 Conclusions

The response agencies have not yet selected remedial actions to address PCB contamination in the Kalamazoo River. Remedial alternatives can cause indirect injuries to natural resources and the services they provide. The Trustees will consider the nature and extent of these indirect injuries as the remedial actions are selected for the site.

9. Conclusions

This document presents the Stage I Injury Assessment of hazardous substance releases, pathways, and injuries in the KRE as a part of the Allied Paper, Inc./Portage Creek/Kalamazoo River Superfund Site NRDA being conducted by federal and state natural resource Trustees. The Stage I Assessment was conducted according to the Stage I Assessment Plan prepared by the Trustees (Michigan Department of Environmental Quality et al., 2000b). The purpose of the Stage I Injury Assessment is to develop preliminary conclusions regarding the nature and extent of natural resource injuries resulting from hazardous substance releases into the KRE. The Stage I Assessment is intended to be preliminary and based primarily on existing data. The results of the Stage I Assessment will be used by the Trustees to help define any additional focused work that could be conducted in the next stage and, if appropriate, to help evaluate any potential settlement options.

Table 9.1 presents a summary of the Stage I Injury Assessment conclusions. For each resource and injury definition, the table presents the conclusions of the Stage I Injury Assessment using the following terms:

- "Yes," meaning that available data demonstrate that the injury in question has occurred
- "No," meaning that available data demonstrate that the injury in question has not occurred
- "Possibly," meaning that there is some available data demonstrating that the injury in question has occurred, but important uncertainties remain
- "Unknown," meaning that the available data are not sufficient to demonstrate that the injury in question either has or has not occurred.

The spatial extent of injuries to resources is described in terms of the KRE Assessment Area, which includes the natural resources within the Portage Creek and Kalamazoo River riparian corridors and in Lake Michigan that are exposed to hazardous substances released from the PRP facilities.

Table 9.1. Summary of Stage I Injury Assessment conclusions

Resource	Injury definition evaluated	Injured?	Spatial extent (if injured)	Notes
Surface water/ sediment	PCBs in excess of drinking water standards	No		
	PCBs in excess of aquatic life standards/criteria	Yes	All of Portage Creek and Kalamazoo River downstream of PRP facilities	
Fish	PCB fish consumption advisories	Yes	Entire assessment area	
	PCBs in excess of FDA tolerance level	Yes	All of Portage Creek and Kalamazoo River downstream of PRP facilities	
	Adverse reproductive effects	Possibly	Only selected Kalamazoo River locations sampled, extent unknown	Uncertainty regarding toxicity benchmarks.
	Sublethal adverse toxicological effects on smallmouth bass	Possibly	Only selected Kalamazoo River locations sampled, extent unknown	Definitive causal link between observed effects and PCBs not established.
	Sublethal adverse toxicological effects on other fish	Unknown		
Benthic invertebrates	Adverse toxicological effects from surface sediment PCBs	Yes	All of Portage Creek and Kalamazoo River downstream of PRP facilities	
Waterfowl	PCBs in excess of FDA tolerance level	Unknown		Limited (and contradictory) data.
Bald eagles	Adverse reproductive effects from PCBs	Yes	Entire assessment area	Two breeding areas in Allegan State Game Area, and one near New Richmond. Depending on suitability of habitat, rest of river downstream of PRP facilities.
Other piscivorous or carnivorous bird species	Adverse reproductive and sublethal effects from PCBs	Possibly	All of Portage Creek and Kalamazoo River downstream of PRP facilities	High PCB exposure, but sensitivity of these birds to PCB toxicity is unknown.

Table 9.1. Summary of Stage I Injury Assessment conclusions (cont.)

Resource	Injury definition evaluated	Injured?	Spatial extent (if injured)	Notes
Passerine birds	Adverse toxicological effects from PCBs	Unknown		Sensitivity of these birds to PCB toxicity is unknown.
Mink	Adverse reproductive effects from PCBs	Yes	All of Portage Creek and Kalamazoo River downstream of PRP facilities	Multiple lines of evidence with consistent conclusions.
Mice, fox	Adverse toxicological effects from PCBs	No		
Muskrat, shrew	Adverse toxicological effects from PCBs	Unknown		Limited data; sensitivity to PCB toxicity is unknown.
Floodplain soils	PCBs in soil predicted to cause adverse effects to biota	Yes	Former impoundments and other areas along the Kalamazoo River	
Molluscs	Adverse community-level effects	Indirect	Impounded areas of the Kalamazoo River	Habitat limited by dams which would otherwise be removed.
Various	Adverse aquatic habitat effects	Indirect	Kalamazoo River areas downstream of former impoundments	Habitat effects from dams that would otherwise be removed.
Various	Indirect injuries from response actions	Unknown (actions not yet selected)	Dependent on location of remedy implementation	Remedy not yet selected, so nature and type of indirect injuries is unknown.

More specifically, the analyses presented in this document support the following Stage I Assessment conclusions:

- PCBs were released from PRP facilities into the KRE.
  - Carbonless copy paper containing PCBs was manufactured from 1954 to 1971. Between 1957 and 1971 over 44 million lbs of PCBs (as Aroclor 1242 and Aroclor 1254) were used in the production of carbonless copy paper across the country. An estimated 20% of this paper (or approximately 8.8 million lbs of PCBs) was recycled at various mills across the country, including those of Allied, Georgia-Pacific, Simpson Plainwell, and Fort James (now owned by Georgia-Pacific) in the KRE.

- The waste stream from PRP facilities was highly contaminated with PCBs. PCBs were detected in effluent and other discharges from facilities, in residuals, and in groundwater. The total contribution of PCBs released by Allied, Georgia-Pacific, Simpson Plainwell, and Fort James facilities into the KRE has been estimated as between 2.2 and 4.4 million lbs (1 and 2 million kg). Releases from PRP facilities are the predominant source of PCBs in the KRE.

- PCBs released from the PRP facilities into the KRE have migrated downstream via surface water pathways. An estimated 36.8 kg (81.1 lbs) of PCBs are contributed annually to Lake Michigan from the Kalamazoo River. Other PCBs have been deposited in instream sediment and in floodplain sediment and soils of Portage Creek and the Kalamazoo River. PCBs are generally persistent in the environment and degrade very slowly.

- KRE surface water has been and is injured as a result of PCB releases.

  - Surface water PCB concentrations downstream of PRP facilities in Portage Creek and the Kalamazoo River exceed applicable water quality criteria established by the State of Michigan and EPA for human cancer risk, and for the protection of aquatic life and piscivorous wildlife. Based on the sources and timing of the releases, it is highly unlikely that PCBs would have been present before the initial release in the mid-1950s and thus it is reasonable to conclude that water did not exceed these criteria before this time. Additionally, the Kalamazoo River and Portage Creek have designated committed uses that are relevant to the exceedence of water quality criteria. The exceedences of relevant criteria and standards occurs throughout the length of Portage Creek and the Kalamazoo River downstream of PRP facilities, and have most likely occurred beginning soon after PCBs were incorporated into the facilities' waste streams.

  - PCB concentrations in surface water are sufficient to cause injury to fish and benthic invertebrates that are exposed to the surface water (described in subsequent bullets).

- KRE sediment has been and is injured as a result of PCB releases.

  - PCB concentrations in surface sediment of Portage Creek and the Kalamazoo River downstream of PRP facilities are nearly three orders of magnitude greater than a consensus-based extreme effect concentration above which toxicity to benthic invertebrates is expected to occur.

- PCB concentrations in surface sediment are sufficient to have caused injury to mink based on food chain dietary exposure. Concentrations are as high as three orders of magnitude greater than a site-specific LOEL for mink.

- Available data indicate that injuries to sediments occur throughout Portage Creek and the Kalamazoo River downstream of PRP facilities.

- KRE fish have been and are injured because of fish consumption advisories issued in response to the PCB releases.

  - PCB fish consumption advisories have been issued since 1979. PCB concentrations in edible tissue for many species have been greater than the trigger levels for advisories as directed by the State of Michigan. Advisories downstream of PRP facilities are more severe and apply to more species than advisories upstream of PRP facilities.

  - PCB concentrations in fish tissue downstream of PRP facilities have also exceeded tolerance levels established by the FDA under the Food, Drug and Cosmetic Act.

  - Areas of fish consumption advisory injuries are the Kalamazoo River from Morrow Dam to Lake Michigan, Portage Creek, and portions of Lake Michigan. Concentrations of PCBs have been sufficient to trigger advisories or exceed tolerance levels since the early 1970s, and exceedences are likely to continue into the future.

- Smallmouth bass in the KRE may be (and may have been) injured as a result of PCB releases.

  - Available data on TCDD-eq concentrations from PCBs in smallmouth bass and walleye eggs generally do not support the conclusion that recent PCB concentrations are sufficient to cause adverse reproductive injuries in these fish. However, a LOEL from the single chronic laboratory exposure study available demonstrate that KRE fish may be suffering from chronic reproductive injuries. The applicability of the chronic exposure study is difficult to assess.

  - PCB concentrations in many KRE smallmouth bass livers are less than available literature toxicity thresholds for sublethal effects. However, few literature thresholds are available, and KRE bass liver PCB concentrations are comparable to concentrations observed in fish from the Lower Fox River/Green Bay (Wisconsin) that had increased incidences of liver tumors and pre-tumors.

- A limited, preliminary study of KRE smallmouth bass showed that bass collected from the assessment area had higher PCB concentrations and significant alterations of body condition, endocrine function, and histopathological status compared to those collected in upstream reference areas. The types of biochemical responses and histopathological observations were consistent with those seen in other sites where freshwater fish have been exposed to PCBs. However, a definitive causal relationship could not be established given the study design.

- Data are not available to evaluate adverse toxicological effects injuries to other fish species.

- Benthic invertebrates have been and are injured by exposure to PCBs in surface sediments that exceed toxicological thresholds for adverse effects. Concentrations in surface sediments from all of Portage Creek and the Kalamazoo River downstream of PRP facilities exceed thresholds.

- It is unknown whether waterfowl are injured due to of exceedences of the FDA tolerance level for poultry because of minimal (and contradictory) available data on PCB concentrations in edible tissue.

- Bald eagles have been and are injured as a result of PCB releases.

  - Bald eagles have dramatically decreased reproductive rates in the Kalamazoo River compared to nesting success in other known PCB contaminated areas and to coastal and inland Michigan averages. The average nesting success between 1990 and 2003 was only 0.2 young per nest, far below normal replacement rates.

  - Failed bald eagle eggs collected in the Kalamazoo River assessment area contain PCB concentrations that are sufficient to cause reproductive effects.

  - Bald eagle nestling plasma samples contain PCB concentrations that are sufficient to cause reproductive effects.

  - The MDEQ ERA concludes that risks to bald eagles from dietary PCB exposure are high. PCB concentrations in KRE fish, a primary bald eagle food item, exceed thresholds for dietary toxicity.

  - Injury to bald eagles dates back to at least 1990, when the first nest was attempted in the Kalamazoo River, and continues to the present day. It is possible that nest failure would have occurred earlier than this, but it appears that no nests were attempted along the Kalamazoo River between 1960 and 1989.

- Other avian species in the KRE may be (and may have been) injured as a result of PCB releases.
  - Eggs of eastern bluebird, great blue heron, house wren, great horned owl, and red tailed hawk contain concentrations of PCBs that exceed relevant toxic thresholds for reproductive effects.
  - Concentrations of PCBs in eggs of various KRE bird species, including mute swan, robin, tree swallow, red-winged blackbird, and wood duck are within or exceed the range associated with embryomortality effects in sensitive species. However, the sensitivity of these species to PCB toxicity is unknown.
  - PCB concentrations in KRE fish exceed thresholds for dietary toxicity to piscivorous birds.
- KRE mink have been and are injured as a result of PCB releases.
  - PCB concentrations in KRE fish exceed dietary thresholds for causing adverse reproductive impacts to mink.
  - PCB concentrations in mink tissue (whole body and liver) exceed concentrations associated with reproductive effects.
  - Mink trapping success is lower in the KRE downstream of PRP facilities compared to a reference location. Although the trapping study was not designed to evaluate mink abundance, the results suggest a lower abundance of mink in PCB contaminated areas.
  - The MDEQ ERA concluded that mink are at risk from dietary exposure pathways.
- Other mammalian species and shrews in the KRE may be (and may have been) injured because of PCB exposure.
  - The MDEQ ERA concluded that other mammalian species such as mice and muskrat are not at risk from dietary exposure pathways. Red fox is unlikely to be at risk from dietary exposure pathways.
  - PCB concentrations in small mammals are lower than or similar to toxicity thresholds for mink, a more sensitive species, and thus small mammals do not appear to be injured.

- PCB concentrations in some shrews are higher than toxicity thresholds for mink. However, the sensitivity of shrews to PCB toxicity is unknown, and thus injury to shrews is uncertain.

- Total PCBs in muskrat livers are elevated downstream of PRP facilities, and concentrations in some samples exceed the reproductive effects TRV for mink. Total PCBs in whole body muskrat do not exceed the whole body reproductive effects TRV for mink. Further analysis would be required to draw conclusions regarding injury to muskrat.

- Floodplain soils in the KRE have been and are injured because of PCB contamination.

  - PCB concentrations in surface floodplain soils exceed site-specific thresholds corresponding with adverse effects to robin and great horned owl via food chain exposure. Exceedences are of the greatest magnitude and frequency in soils of the former impoundments.

- Indirect injuries to resources have been occurring and may continue to occur as a result of response actions taken to address PCB contamination in the KRE.

  - Because of the presence of PCBs in sediments stored behind them, several dams along the Kalamazoo River that were targeted for removal have remained in place. As a result, the aquatic habitat has been injured. Mussel populations are adversely affected in areas behind the dams, and other resources have been affected as well.

  - Future indirect injuries may be caused by PCB response actions. At this time, remedial actions for the Kalamazoo River have not been selected by the response agencies. The nature and extent of indirect injuries and natural resource service losses will vary depending on the remedial actions that are implemented. The Trustees are coordinating with the EPA and MDEQ Superfund Section to minimize indirect injuries associated with the remedial options and will consider any indirect injuries associated with remedial actions in future stages of the NRDA.

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